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OBSERVATIONS ON THE TOXIC EFFECTS OF CORDITE

BY

J. S. WEINER and M. L. THOMSON

From the Royal Naval Personnel Research Committee of the Medical Research Council

(RECEIVED FOR PUBLICATION, APRIL, 1947)

The main object of the present trials was to ascertain whether ill effects attributable to cordite could be reproduced experimentally. Additional aims were to obtain information on (1) the mode of absorption of cordite, (2) the influence of cordite fumes on the efficiency of operatives at ambient and at high wet-bulb temperatures, (3) the occurrence of any acquired immunity, (4) the extent, if any, to which carbon dioxide might contribute to the symptoms, and (5) the concentration of nitrogen dioxide and carbon monoxide released during cordite handling in addition to the vapour of nitroglycerine.

Methods

Trial Room.—A trial room (fig. 1, p. 230) was erected in a building provided with steam and dry heat. The plan (fig. 2) shows the dimensions of the two parts of the airtight room—(1) the space (380 cu. ft.) in which four subjects and two observers were accommodated, and in which the cordite was handled and (2) the stowage section at one end, in which twenty cordite cases were stacked in five horizontal rows of four each (fig. 3). In the hot experiments the trial room was heated by raising the temperature of the outer room. Some control over the dry-bulb temperature was obtained by regulating the steam heating and by spraying the metal roof of the trial room, on to which a fan was directed.

Subjects.—The subjects were eight healthy young men of aged 20 to 25 years, who were medically examined and had their chests radiographed before leaving barracks. They were arranged in two teams of four, each team subdivided into two pairs.

Clothing.—Clothing consisted of a single-piece overall worn over a pair of shorts, anti-flash helmet (with mask), anti-flash gloves, socks, and plimsolls. One member of each pair wore in addition a rubber apron, with full length sleeves, extending from neck to knees. The anti-flash gloves were replaced by rubber gauntlet gloves into which the sleeves of the apron were tucked. This dress protected the wearer's skin from contact with the cordite, and was worn by each man on alternate days.

Cordite.—The cordite was packed in metal cases containing two charges each of 96 lb. weight. It had been stoved for five months at 120° F. Each charge consisted of many cylindrical cordite rods (average diameter 0·2 inches) enclosed in a silk bag.

Analysis of carbon dioxide, nitrous fumes (nitrogen dioxide), and carbon monoxide of the air within these cases revealed marked variations from case to case. Since full analysis of the gases was possible in a limited number only, the cases were arranged on the basis of their carbon dioxide content, as this was found to show a fair positive relationship to the carbon monoxide content, but no correlation with the nitrogen dioxide content. The cases to be opened were then preselected so as to give, as far as possible, an equal proportion of these gases during each trial and also during each work period.

Dummy Charges.—These were intended to be replicas of the cordite charges. They weighed 108 lb. (against 96 lb. for the cordite charges) and were packed in pairs in the ordinary cases. The fabric surrounding the dummy was, however, different from that of the cordite charges, and it was not difficult for the men to detect which of the two they were handling.

Observations

Air Analysis.—Samples of air taken from the cordite cases, as well as from the experimental room at intervals during the trials, were analysed for nitrogen dioxide by the Greiss-Ilosvay method, for carbon monoxide by the iodine-pentoxide method, and for oxygen and carbon dioxide by Haldane and Orsat gas analysis apparatus. Attempts to estimate nitro-glycerine (N.G.) vapour in the presence of the much higher nitrogen dioxide content were unsuccessful.

Physiological.—The men were weighed nude at the beginning and again at the end of each trial. Their net body-weight loss was determined, making allowance for water drunk and urine output. Water was allowed in any quantity. The clothing, including rubber protective garments when worn, was weighed at the beginning and at the end. Pulse rates were counted in the standing position before commencing work and in the first half minute after stopping work. Respiratory rate was counted in the standing position before work and in the second minute after stopping work. Blood pressures were measured in every case after a preliminary period in which the subject sat quietly for five minutes. The blood pressures in the standing position were taken one minute after the erect posture was assumed. Rectal temperatures were measured before and after each work bout.

Fitness Index.—An assessment of the ease with which the man performed the work was attempted by questioning the subject, and from the observer's opinion of his condition. The following rating was used: *Grade* 1, perfectly able to continue work; *Grade* 2, able to finish work but doubtful if a second bout could be done efficiently; *Grade* 3, able to finish work, but work done inefficiently, and/or work followed by symptoms of faintness; *Grade* 4, able to finish work but work done inefficiently and followed by collapse; or unable to finish work.

Subjective Reactions.—The men were questioned regarding their general condition with a view to ascertaining the incidence, if any, of headache. No leading question on this point was asked, but after the first cordite trials implications of this questioning were, of course, appreciated by the men.

Body Fluid Estimations.—A few preliminary estimations of whole blood nitrites were made. Methæmoglobin concentration was estimated on one occasion by Dr. Jope of the London Hospital. Blood examinations and urine analyses were carried out by Dr. R. Case, of the Royal Naval Physiological Laboratory.

Routine of Trials.—The duration of each trial was three hours. Each couple performed two twenty-minute periods of work at the commencement and at the end of every trial. During each work period in cordite trials, four cases were opened and the cordite charges carried to the racks (fig. 2) and then replaced in the cases. Thus sixteen fresh cases of cordite were opened each day.

Blood pressure, rectal temperature, and pulse were taken before and after each work period except in the case of protected men where blood pressure readings had to be omitted after the first and before the second work.

The atmosphere of the trial room was sampled on entry and at the end of the first, second, and third hours.

The procedure on Dummy trial days was identical except that the cordite cases were replaced by dummy cases. The programme of trials is shown in Table 1.

As can be seen, the experiments were arranged so that (*a*) dummy experiments preceded and followed the two cordite trials in both 'hot' and 'cold' series, to allow for any effect of repetition in improving the performance, especially at high temperatures, and (*b*) three clear days, with one exception, were allowed between successive exposures to cordite.

Results

Observations on "Aged" Cordite Products.—Estimations of carbon dioxide were carried out on the gas within each of the 128 cases used; nitrogen dioxide and carbon monoxide were determined in 18 cases and oxygen in 7 cases only. A marked variation was found from case to case in the concentrations of these gases, ranging from 0·10 per cent. to 24 per cent. in the case of carbon dioxide; from 4·5 per cent. to 20·8 per cent. of oxygen, and from 0·00005 to 0·6 per cent. of carbon monoxide.

There was an inverse relationship between the oxygen and carbon dioxide contents of each of the 7 cases. There was a fair relationship between carbon dioxide and carbon monoxide contents; carbon dioxide and nitrogen dioxide concentrations did not appear to be related.

The concentration of nitroglycerine vapour could only be inferred from its known vapour pressure. It was presumably the same in all of the cases, and was calculated to be of the order of 0·0000003 per cent. W/V during "cold" weather trials.

The dead space in each case is about 20 litres, and 16 were opened during each trial, so that 320 litres of case air were diluted by 10,000 litres of room air (31x).

The cases were so constructed as to be flashtight. It was noticed, however, during sampling, that the contents were invariably at or about atmospheric pressure under temperature changes which involved expansion and contraction of the gases, and further tests by manometer confirmed that few, if any, of the cases were airtight.

A few samples were taken during hot trials close to the case as the charge was being withdrawn during work. Nitrogen dioxide was 0·00012 per cent. to 0·0004 per cent. and carbon monoxide a 'trace' to 0·002 per cent. These figures are unlikely to represent the maximum concentrations to which the men were exposed while withdrawing charges.

A pair of anti-flash gloves was analysed for nitroglycerine after the second cold cordite (May 7, 1946) and again after the second hot cordite (June 4, 1946) trials. The average weight of each glove was 50 g. On May 7, 1946, weight of nitroglycerine in the left glove was 0·0680 g., and of the right glove 0·0599 g. On June 4, 1946, the

TABLE 1

PROGRAMME OF TRIALS

Date	Experiment	Conditions	Team
April, 1946			
Mon. 29	Dummy	Cold	B
Tues. 30	,,	,,	A
May, 1946			
Thur. 2	Cordite	,,	B
Fri. 3	,,	,,	A
Mon. 6	,,	,,	B
Tues. 7	,,	,,	A
Thur. 9	Dummy	,,	B
Fri. 10	,,	,,	A
Mon. 13 to Sat. 18	First week of acclimatization		
Mon. 20 to Sat. 25	Second week of acclimatization		
Mon. 27	Dummy	Heat	B
Tues. 28	,,	,,	A
Thur. 30	Cordite	,,	B
Fri. 31	,,	,,	A
June, 1946			
Mon. 3	,,	,,	A
Tues. 4	,,	,,	B
Thur. 6	Dummy	,,	A
Fri. 7	,,	,,	B

weight of nitroglycerine in the left glove was 0·0177 g. and in the right glove 0·0045 g. These amounts of nitroglycerine were probably concentrated in the palms of the gloves and anterior surfaces of the gauntlets.

Environmental Observations

Concentrations of NO_2 and CO in the Trial Room.—With few exceptions nitrogen dioxide and carbon monoxide were maximal in the terminal sample (Tables 2 and 3). The final nitrogen dioxide figures were in cold trials from 0·0002 per cent. to 0·0007 per cent., and in hot trials from 0·0001 per cent. to 0·0004 per cent.; corresponding carbon monoxide figures were 0·001 per cent. to 0·002 per cent. in cold trials and "traces" to 0·001 per cent. in hot trials.

The average of all analyses of nitrogen dioxide for "cold" trials was 0·00032 per cent. and for hot trials was 0·00015 per cent.; corresponding figures for carbon monoxide were 0·0011 per cent. and 0·00057 per cent. From these figures it can be seen that the average concentration of these gases was twice as high during exposure to cold temperature as it was during hot trials.

TABLE 2

ATMOSPHERIC CONDITIONS DURING TRIALS IN TEMPERATE ENVIRONMENTS

Team A (4 subjects)

Date and trial	Dry bulb (av. ° F.)	Wet bulb (av. ° F.)	Air concentrations (at end)		
			CO_2 %	NO_2 %	CO %
30.4.46 Dummy	75·6 (71·5-79·0)	68·2 (65·5-75·0)	3·6		
2.5.46 Cordite	71·6 (65·0-75·0)	66·4 (60·8-70·0)	4·0	0·00020	0·0014
6.5.46 Cordite	67·9 (60·0-71·5)	62·8 (55·6-66·5)	3·6	0·00067	0·0008
9.5.46 Dummy	72·2 (65·2-74·0)	64·9 (59·6-68·5)	3·2	0·00025	0·0002

Team B (4 subjects)

29.4.46 Dummy	70·5 (65·0-72·5)	65·4 (60·0-67·5)	3·1		
3.5.46 Cordite	71·2 (67·5-74·0)	67·0 (59·0-69·5)	4·1	0·00021	0·0018
7.5.46 Cordite	69·2 (65·0-74·0)	64·6 (59·0-69·5)	4·0	0·00045	0·0020
10.5.46 Dummy	72·3 (64·8-76·9)	66·6 (59·2-71·3)	3·6		

TABLE 3

ATMOSPHERIC CONDITIONS DURING TRIALS IN HOT ENVIRONMENTS

Team A (4 subjects)

Date and trial	Dry bulb (av. ° F.)	Wet bulb (av. ° F.)	Air concentrations (at end)		
			CO_2 %	NO_2 %	CO %
28.5.46 Dummy	89·0 (88·4-89·5)	86·7 (81·5-88·2)	4·2		
31.5.46 Cordite	88·6 (88·0-89·0)	86·6 (79·5-88·2)	4·2	0·00037	Traces
3.6.46 Cordite	88·5 (85·5-90·5)	86·2 (77·8-89·2)	4·3	0·00012	0·0010
6.6.46 Dummy	88·3 (87·0-89·0)	85·6 (79·8-85·6)	—	0·00009	Traces

Team B (4 subjects)

(24.5.46 Dummy preliminary)	91·8 (90·0-92·6)	90·7 (85·8-92·3)	3·8		
27.5.46 Dummy	87·3 (85·8-88·8)	84·3 (79·0-86·1)	—	0·00021	Traces
30.5.46 Cordite	90·3 (88·0-93·0)	87·8 (81·5-92·0)	4·0	0·00026	0·0006
4.6.46 Cordite	87·35 (87·0-89·0)	84·1 (79·2-87·0)	4·1	0·00018	0·0008
7.6.46 Dummy	90·95 (88·5-91·2)	87·4 (77·5-90·5)	3·6		

It is also interesting that in the dummy trials in which air was analysed the nitrogen dioxide and carbon monoxide concentrations were of the order found in some of the hot cordite trials. This may indicate the difficulty of ensuring the complete absence of cordite gases from the vicinity.

CO_2 Concentrations.—The concentrations reached at the end of the trials are shown in Tables 2 and 3. These are, physiologically speaking, quite high, but it must be remembered that concentrations over 3 per cent. were only breathed during the last hour of the experiment. It will be noted that the carbon dioxide percentages are, with two exceptions, lower at the end of dummy trials than at the end of cordite trials. The probable explanation is that the two observers had a good deal more physical exertion to perform during the cordite runs, in taking off the lids which were often almost immovable, and in groping for and extracting a wooden disc from the

far end of the cylinder. As is reported below, the oxygen cost of the work was not substantially different, whether dummy or cordite charges were handled.

Thermal Conditions.—During exposure at temperate conditions (Table 2) the dry-bulb and wet-bulb temperatures rose rapidly in the first half hour by 2° to 5° F. in different experiments, and then more slowly in the remaining hour and a half—a further 3° to 6° F. In some of the high-temperature experiments (Table 3) the rise in dry-bulb temperature was only 2° F. throughout, owing to the improved control measures which were instituted as a result of experience. The wet-bulb, however, as in the cooler conditions, rose rapidly in the first twenty to thirty minutes and then more slowly. It should be noted that during the most important part of the exposure, namely, for three out of the four work periods performed by the two teams, the temperature rise was only about 2° F. in dry bulb or wet bulb. At high temperatures this increase means, of course, that the second work was done at a wet-bulb temperature physiologically appreciably more severe than that of the first work spell.

Table 2 shows that both teams performed one cordite trial at temperatures a little below either of the dummy runs. On the whole the differences between the series of dummy and cordite trials have not been regarded as significant from the view point of the present trials. The results of the temperate trials of both teams have accordingly been treated together.

In the case of the trials at high temperatures it has been thought better, for the most part, to treat the results for the two teams separately. As Table 3 indicates, the thermal conditions were far more comparable in the case of team A than in the case of team B. For the latter it is possible to compare equivalent sets of conditions by taking the first dummy trial in relation to the second cordite trial and the second dummy trial in relation to the first cordite trial. This procedure to some extent tends to equalize the differences in acclimatization between the two pairs; fortunately the pairs are comparable also, in that the same sets of clothing were worn by the same individuals. It may be said at once that in spite of the divergences of treatment undergone by the two teams, the main results are in most particulars very similar.

Estimations of the air movement carried out in five points in the room, both when the men were resting and when they were working, revealed very low air speeds, viz., 8 feet per minute at rest, 20 feet per minute when at work. The work created little air movement because each journey was so short and so infrequent.

Oxygen Cost of the Work.—There was a marked difference in weight between the dummy and cordite charges, namely, 108 lb. as compared to 96 lb. On this account any of the effects of cordite would have been under-estimated. On the other hand, the "aged" cordite coverings were sticky, and this made it rather more difficult to remove the charges from the cylinders. In the event the actual determinations of oxygen consumption gave a result very similar for the two types of charge; the cost of twenty minutes' handling of either cordite or dummy, followed by thirty minutes' recovery, was, for a subject (CO) well practised in the method, 240 kg. calories per hour. The oxygen cost of the work itself, measured after a preliminary ten minutes work, was found to be 310 kg. calories per hour. Wearing the rubber gloves and apron in the cold did not increase the oxygen cost appreciably.

Physiological Findings

Subjective Effects.—On cold cordite trials every person taking part experienced irritation of the nose and throat, which caused intense congestion of the nasal mucous membrane, sneezing, and coughing. This passed off, but was noticed again every time a fresh batch of cases was opened. This irritant effect was less noticeable on hot cordite days.

Tables 4 and 5 set out the incidence of headaches during and after the work routine throughout the experimental series.

At ambient air temperatures the Tables show that headaches were complained of both by the observers and by subjects handling cordite. Headaches were noticed during the first work spell in some cases, but were reported on more frequently after the commencement of the second spell. On the first cordite day, at ambient temperatures, five of the eight subjects reported symptoms during the second work, and, after leaving the experimental room, six of the eight reported headaches. The two subjects who did not complain, namely CO of team A and JA of team B, appeared to be consistently immune in this respect, both in these trials and in those at high temperatures. A comparison of the second cordite day with the first cordite day at both temperatures with regard to incidence of headaches suggests that some immunity or tolerance was acquired; the intensity of the headache was also, on the whole, less severe.

During the two days when cordite was handled in hot surroundings, headaches were reported during exposure by the subjects of team B only; one subject of team A complained of headache only after leaving the room. The observers of both teams complained of headaches on six out of eight exposures. In all, seven out of twelve individuals exposed suffered headaches on the first cordite day and five out of twelve on the second. The incidence in hot conditions appears to be less than in the cold series. The character of the headache was similar, but on the whole less severe.

The outstanding characteristics of the headache were its severity and its frontal situation (which was invariably reported). With time the headache often spread to the vertex. Unlike some reports in the literature, the headache was never described as "throbbing," though one individual said it "lifted the top of his head off." In many cases the headache passed off for a while during the trial, but returned later, usually on leaving the trial room, and persisted a variable time up to twenty-four hours. When severe there were symptoms of nausea, and in several cases there were complaints of abdominal pain.

Fitness Rating.—Tables 4 and 5 show the fitness ratings assessed before and after each work throughout the

experimental series. In temperate environments cordite handling was performed, according to the subjects, " not quite so easily as in dummy runs." This opinion was not expressed only by those men with headaches. However, we do not consider that at these temperatures the men were demonstrably affected as regards performance.

At high temperatures the picture was very different. On both occasions when cordite was handled by team A at high wet-bulb temperatures, the ability to carry out the second bout of work was impaired. Although the work spell was completed in every case (with one exception) the condition of the men during and after the work was much poorer than on dummy days. As indicated in Table 5, the men came near to collapse on half the occasions during cordite trials, with pallor, dizziness, and feelings of faintness.

In the case of team B essentially similar results were found. At the higher wet-bulb temperature (87° F.) one of the four men came near to fainting after the second bout of work. At the lower wet-bulb temperature (84° F.) although there was no collapse, the men were undoubtedly in a worse state than on the corresponding dummy day.

Blood Pressure.—The effects on the blood pressure of cordite handling in ambient temperatures is shown in Table 6, which gives the averages for all eight subjects, sitting and standing ; the " protected " (P) and " unprotected " (U) findings are shown separately. The effect of cordite, as can be seen, is to produce a consistently greater fall in systolic blood pressure (S.B.P.) than occurs in dummy experiments. This effect was shown on eight out of ten occasions sitting, and on nine out of ten occasions standing.

The high temperature results are shown in Tables 7A and 7B, for teams A and B respectively. As in the cold, cordite produced a specific increase in the fall of S.B.P. ;

TABLE 4

EFFECT ON SUBJECTIVE REACTIONS AND WORK PERFORMANCE OF CORDITE AND DUMMY HANDLING IN TEMPERATE ENVIRONMENTS

Subject	First cordite day					Second cordite day				First dummy day		Second dummy day	
	Protection	1st Work	2nd Work	After exit	Protection	1st Work	2nd Work	After exit		Work		Work	
		H I	H I	H		H I	H I	H		1st	2nd	1st	2nd
										I	I	I	I
Team A													
MU	U	− 1	+ 1½	++	P	− 1	+ ½	++		1	1½	1	1
CO	P	− 1	− 1½	−	U	− 1	− ½	−		1	1	1	1
WH	P	− 1	+ 1½	++	U	− 1	− ½	−		1	1	1	1
MA	U	− 1	+ 1½	+	P	− 1	− 1½	−		1	1	1	1
Observers													
TH		+	++	++		+	+	++					
TI		+	+	++		+	+	++					
Team B													
MC	U	− 1	− 1½	+	P	+ 1	+ 1½	+		1	1½	1	1
COE	P	+ 1	++ 1½	++	U	− 1	+ 1½	+		1	1½	1	1
OR	P	++ 1	++ 1½	++	U	+ 1	+ 1½	++		1	1	1	1
JA	U	− 1	− 1	−	P	− 1	− 1	−		1	1	1	1
Observers													
WE		+	+	++		+	+	+					
TI		+	+	++		+	+	+					
Proportion of subjects with headaches		2/8	5/8	6/8		2/8	4/8	4/8					
Proportion with headaches (subjects and observers)		6/12	9/12	10/12		6/12	8/12	8/12					
Average fitness index for subjects		1	1·5			1	1·5			1	1·2	1	1

U = anti-flash without protective garments.
P = anti-flash with protective garments.
H = headache complained of : + = moderate.
++ = severe.
I = fitness index.

that is to say, over and above the fall due to heat alone. The proportion of observations in which this fall was demonstrated was fourteen out of sixteen (sitting) and fifteen out of sixteen (standing).

This effect of cordite has been analysed statistically (Table 8) by taking results comparable as regards clothing, the individual, and the temperature conditions. The mean difference between the change in S.B.P. in the dummy and cordite trials respectively, as compared with its standard deviation, gives a 't' test result of P=0·05 to 0·02 (sitting and standing) in temperate conditions, and P=0·001 (sitting and standing) in hot conditions.

The probability, therefore, of the present findings occurring as the result of random variations is small.

A more detailed analysis of the data for the two teams, both in the cold and in the heat, revealed no regular difference between protected and unprotected clothing.

The diastolic blood pressure almost invariably rose during the trials. Tables 6, 7A, and 7B also show that the pulse pressure nearly always diminished more during cordite trials than during dummy trials.

Pulse and Respiratory Rates (Tables 6, 7A, and 7B).—Wearing the protected clothing increased the pulse rate in both the hot and the cold trials. The effect of cordite was not consistent throughout. In the cold the average increase in pulse rate was greater as the result of handling cordite. This was shown also in the heat by team A, but not by team B.

The respiratory rate increased more in the cordite trials than in the dummy trials. This was possibly due to the somewhat higher carbon dioxide concentration attained during the cordite experiments (Tables 2 and 3).

TABLE 5

EFFECT ON SUBJECTIVE REACTIONS AND WORK PERFORMANCE OF CORDITE AND DUMMY HANDLING IN HOT ENVIRONMENTS

Subject	First cordite day					Second cordite day					First dummy day		Second dummy day	
	Protection	1st Work		2nd Work		After exit	Protection	1st Work		2nd Work		After exit	Work 1st	Work 2nd	Work 1st	Work 2nd
		H	I	H	I	H		H	I	H	I	H	I	I	I	I
Team A																
MU	U	−	1½	−	4	+	P	−	1	−	3½	−	1	1½	1	1½
CO	P	−	1½	−	3	−	U	−	1	−	1½	−	1	1	1	1½
WH	P	−	1½	−	3	−	U	−	1	−	3½	−	1	1½	1	1½
MA	U	−	1	−	4	−	P	−	1	−	3½	−	1	1½	1	1½
Observers																
TH		+		+		++		+		++		++				
KI		++		++		++										
PA								−		−		−				
Team B																
MG	U	+	1	+	−*	+	P	−	1	+	2½	+	1	1	1	1
COE	P	−	1	+	3	+	U	−	1	+	2	+	1	1	1	1½
OR	P	+	1	+	2	+	U	−	1	+	2	+	1	1	1	1½
JA	U	−	1	−	1	−	P	−	1	−	1	−	1	1	1	1
Observers																
WE		+		+		++		+		+		+				
PA		+		+				−		−		−				
Proportion of subjects with headaches		2/8		3/8		4/8		0/8		3/8		3/8				
Proportion with headaches (subjects and observers)		6/12		7/12		7/12		2/12		5/12		5/12				
Average fitness index for subjects			1·2		2·8				1		2·4		1	1·2	1	1·4

U=anti-flash without protective clothing.
P=anti-flash with protective garments.
I=fitness index.
H=headache complained of: + =moderate.
++=severe.

* Injured finger, fainted, and was unable to do work.

Rectal Temperatures.—There was no difference in rectal temperatures between cordite and dummy trial days. The rise in rectal temperature as the result of the two work spells in the heat, though small, was little higher in the case of protective garments. The slight rise in rectal temperature was to be expected as the result of the moderate rate of the work.

Weight Loss.—No constant difference was found as the result of cordite. As was to be expected, the protective clothing increased the weight loss. The rate of sweating as shown in the Tables was not very high in relation to the high wet-bulb temperature and still air conditions, pointing again to the relatively low rate of work. It is possible, of course, that incomplete acclimatization may also have been a factor. A comparison on this score between the first and last dummy experiments in the heat, as well as between the first and second cordite days (Table 1) does not, as far as the data permit, support the latter suggestion.

Unevaporated Sweat.—As expected, there was a greater amount of sweat in the clothes when wearing protective garments, but cordite did not produce a consistent effect.

Blood and Urine Tests : Radiograph of Chest.—The blood of one of the authors was submitted to spectrographic analysis three and a half hours after the end of a cordite trial, while he still had a severe headache, by Dr. Jope of the London Hospital. It was found to contain less than 0·2 per cent. of methæmoglobin and to be completely normal in this respect.

TABLE 6

PHYSIOLOGICAL EFFECTS OF CORDITE AND DUMMY HANDLING IN TEMPERATE CLIMATES : DUMMY TRIALS (TWO DAYS)

	First work				Second work				Col. 1 vs. Col. 4		Col. 2 vs. Col. 4	
	Before		After		Before		After					
	U	P	U	P	U	P	U	P	U	P	U	P
	(1)		(2)		(3)		(4)		(5)		(6)	
B.P. Sitting												
Sys. P.	110·0	110·0	110·5		106·0		107·9	101·0	− 2·1	− 9·0	− 2·6	
Dias. P.	60·0	63·0	64·3		65·9		65·3	66·0	+ 5·3	+ 3·0	+ 1·0	
Pulse P.	45·4	47·0	46·2		40·1		42·6	35·0	− 7·4	−12·0	− 3·6	
B.P. Standing												
Sys. P.	120·3	123·0	119·0		116·7		119·0	115·0	− 1·0	− 8·0	− 0·0	
Dias. P.	80·3	77·5	80·8		82·4		88·3	79·0	+ 8·0	+ 1·5	+ 7·5	
Pulse P.	40·0	45·5	38·2		34·3		30·7	36·0	− 9·3	− 9·5	− 7·5	
Pulse rate per minute Standing	80	85		119	80	83	106	108	+26	+23		
Respiratory rate per minute	20	20	25	25	20	20	25	24	+ 5	+ 4		
Rectal T. (°F.) ..	98·9	98·7	98·9	99·2	98·2	9·2	98·6	98·9	− 0·3	+ 0·2	− 0·3	+0·3
	Cordite Trials (2 days)											
B.P. Sitting												
Sys. P.	109·0	108·0	109·0		100·5		93·5	94·7	−15·5	−18·3	−15·5	
Dias. P.	62·3	60·3	66·2		64·8		65·3	72·0	+ 3·0	+11·7	− 0·9	
Pulse P.	46·7	47·7	42·8		35·7		28·2	22·7	−18·5	−25·0	−14·6	
B.P. Standing												
Sys. P.	125·0	118·3	116·8		107·8		102·5	103·8	−22·5	−14·5	−14·3	
Dias. P.	83·0	77·0	83·0		82·8		80·3	83·2	− 2·7	+ ·2	− 2·7	
Pulse P.	42·0	41·3	33·8		25·0		22·2	20·6	−19·8	−20·7	−11·6	
Pulse rate per minute Standing	89	87	123	117	90	92	118	120	+29	+33		
Respiratory rate per minute	19	18	25	28	21	20	25	28	+ 6	+10		
Rectal T. (°F.) ..	98·6	98·5	98·9	98·8	98·1	97·9	98·6	98·4	− 0·0	− 0·1	− 0·3	−0·4

U = anti-flash without protective clothing.
P = anti-flash with protective clothing.

Estimations of nitrite in the blood were inconclusive. The hæmatological and urine analyses carried out by Dr. Case showed no definite abnormality.

Six of the subjects had a radiographic examination of the chest before commencement of the trials, and again on Wednesday, May 6, 1946, when from one to two days had elapsed since the last exposure to cordite. No abnormality was found.

Acclimatization.—It had been planned that the cold trials were to be followed by two weeks of intensive acclimatization. Owing to work on the steam-heating system of the experimental room, the first five of the ten acclimatization days were carried out in another chamber, where it was not found possible to raise the wet bulb above 75° F. Dry bulbs of 90° F. on the first two days and 115° F. on the next three days were attained. As the air movement was high, the conditions were not as severe as those during trials. The next five days of acclimatization were carried out in conditions resembling the experimental series and included two preliminary "dummy" trials, one for each team, inside the experimental room; these were done at a wet bulb at nearly saturated conditions and were more severe than any of the trials.

It is important to note that in these two severe trials the teams were able to complete the experimental routine remarkably well. This indicates that the men had acquired a certain level of acclimatization in spite of the shortcomings of the programme.

TABLE 7A

PHYSIOLOGICAL EFFECTS OF CORDITE AND DUMMY HANDLING AT HIGH TEMPERATURES: TEAM A

	First work				Second work				Col. 4 vs. Col. 1		Col. 4 vs. Col. 2
	Before		After		Before		After				
	U	P	U	P	U	P	U	P	U	P	U
	(1)		(2)		(3)		(4)		(5)		(6)
DUMMY TRIALS (2 days) (each figure represents 4 observations)											
B.P. Sitting											
Sys. P.	103·0	106·5	101·5		98·8		100·5	95·0	− 2·5	−11·5	− 1·0
Dias. P.	61·5	61·5	69·0		61·0		67·5	62·5	+ 6·0	+11·0	− 1·5
Pulse P.	41·5	45·0	32·5		37·0		33·0	32·5	− 8·5	−12·5	+ 0·5
B.P. Standing											
Sys. P.	111·5	114·5	101·5		94·0		100·0	102·5	−11·5	−12·0	− 1·5
Dias. P.	75·0	69·0	74·0		75·0		76·8	80·0	+ 1·8	+11·0	+ 2·8
Pulse P.	36·5	45·5	27·5		19·0		23·2	22·5	−13·3	−23·0	− 4·3
Pulse Rate Standing	84·0	97·0	123·0	125·0	77·0	84·0	114·0	160·0	+30·0	+63·0	
Respiratory rate (min.)	20·0	22·0	30·0	23·0	22·0	22·0	30·0	29·0	+10·0	+ 7·0	
Rectal temp. (°F.)	99·3	99·5	99·9	100·2	99·6	99·9	99·9	100·5	+ 0·6	+ 1·0	
Net weight loss (g.)	—	—	—	—	—	—	—	—	931	1354	
Sweat in clothes (g.)	—	—	—	—	—	—	—	—	487	791	
CORDITE TRIALS (2 days) (each figure represents 4 observations)											
B.P. Sitting											
Sys. P.	102·5	105·5	84·0		82·0		83·5	81·3b	−19·0	−24·2	− 0·5
Dias. P.	53·3	60·5	71·0		60·5		58·5	61·3	+ 5·2	+ 0·8	−12·5
Pulse P.	49·2	45·0	13·0		22·5		25·0	20·0	−24·2	−25·0	+12·0
B.P. Standing											
Sys. P.	111·0	113·5	97·0		81·3a		87·8	81·3b	−23·2	−32·2	− 9·2
Dias. P.	79·0	75·5	78·5		64·0		74·0	66·0	− 5·0	− 9·5	− 4·5
Pulse P.	32·0	38·0	18·5		17·3		13·8	15·3	−18·2	−22·7	− 4·7
Pulse Rate Standing	88·0	87·0	121·0	150·0	97·0		132·0	164·0	+44·0	+77·0	
Respiratory rate (min.)	19·0	20·0	30·0	29·0	22·0	21·0	30·0	29·0	+11·0	+ 9·0	
Rectal temp. (°F.)	99·5	99·3	99·4	100·0	99·7	99·7	100·1	100·3	+ 0·6	+ 1·0	
Net weight loss (g.)	—	—	—	—	—	—	—	—	733	1326	
Sweat in clothes (g.)	—	—	—	—	—	—	—	—	417	579	

a=1 subject 'unprotected' collapsed. 3 figures only obtained.
b=1 subject 'protected' collapsed. 3 figures only obtained.
U=Anti-flash without protective garments. P=Anti-flash with protective garments.

Discussion

The experiments detailed above make clear the detrimental effects of handling cordite as compared with handling dummies, both in cool and in hot humid environments. As already mentioned the dummy bags were easily distinguished from the cordite bags; owing to the particular circumstances of the investigation the difficulties in making the bags really comparable could not be overcome. Thus, after stoving—the necessary preliminary to these experiments—the fabric surrounding the cordite charges became discoloured, corroded, and rather friable. To simulate this covering on the dummies would have required the provision of an identical covering, using perhaps bags in which cordite had previously been stored after all traces of nitroglycerine and their products had been removed. Again, after stoving, the characteristic smell of the cordite would need to be simulated, also a very difficult task.

These differences between the dummy and cordite days must of course be borne in mind in assessing the significance of the subjective effects associated specifically with cordite handling. Apart from the

TABLE 7B

PHYSIOLOGICAL EFFECTS OF CORDITE AND DUMMY HANDLING IN HIGH TEMPERATURES: TEAM B

	First work				Second work				Col. 4 vs. Col. 1		Col. 4 vs. Col. 2
	Before		After		Before		After				
	U	P	U	P	U	P	U	P	U	P	U
	(1)		(2)		(3)		(4)		(5)		(6)
DUMMY TRIALS (2 days) (each figure represents 4 observations)											
B.P. Sitting											
Sys. P.	104·0	102·0	99·0		90·0		92·3	94·3	−11·7	− 7·7	− 6·7
Dias. P.	58·0	60·0	62·0		61·0		62·3	63·5	+ 4·3	+ 3·5	+ 0·3
Pulse P.	46·0	42·0	37·0		29·0		30·0	30·8	−16·0	−11·2	− 7·0
B.P. Standing											
Sys. P.	112·5	110·5	108·0		98·0		101·0	102·5	−11·5	− 8·0	− 7·0
Dias. P.	75·0	69·3	76·5		74·0		78·0	77·5	+ 3·0	+ 8·2	+ 1·5
Pulse P.	37·5	41·2	31·5		24·0		23·0	25·0	−14·5	−16·2	− 8·5
Pulse Rate Standing	82·0	83·0	112·0	149·0	91·0	82·0	110·0	146·0	+28·0	+63·0	
Respiratory rate (min.) ..	21·0	22·0	27·0	28·0	25·0	24·0	30·0	32·0	+ 9·0	+10·0	
Rectal temp. (°F.)	98·9	98·9	99·6	99·4	99·1	98·9	99·5	99·6	+ 0·6	+ 0·7	
Net weight loss (g.)	—	—	—	—	—	—	—	—	595	1099	
Sweat in clothes (g.)	—	—	—	—	—	—	—	—	375	529	
CORDITE TRIALS (2 days) (each figure represents 4 observations)											
B.P. Sitting											
Sys. P.	107·3	106·0	94·0		92·5		86·5	84·0	−20·8	−22·0	− 7·5
Dias. P.	57·8	59·5	54·0		56·7		60·0	67·0	+ 2·2	+ 7·5	+ 6·0
Pulse P.	49·5	46·5	40·0		35·8		26·5	17·0	−23·0	−29·5	−13·5
B.P. Standing											
Sys. P.	116·5	113·5	104·5		102·3		95·5	91·0	−21·0	−22·5	− 9·0
Dias. P.	74·5	71·0	76·5		75·0		70·5	*77·0	− 4·0	*+ 6·0	− 6·0
Pulse P.	42·0	42·5	28·0		27·3			*14·0	−17·0	*−28·5	− 3·0
Pulse Rate Standing	84·0	82·0	108·0	133·0	93·0	99·0	111·0	142·0	+27·0	+60·0	
Respiratory rate (min.) ..	21·0	20·0	28·0	24·0	24·0	24·0	31·0	37·0	+10·0	+17·0	
Rectal temp. (°F.)	98·8	98·8	99·6	99·9	99·2	99·2	99·8	100·8	+ 1·0	+ 1·2	
Net weight loss (g.)	—	—	—	—	—	—	—	—	791	1093	
Sweat in clothes (g.)	—	—	—	—	—	—	—	—	461	746	

Two observations only.

U = Anti-flash without protective garments. P = Anti-flash with protective garments.

fact that these particular effects were noted by the observers as well as the subjects and were of a characteristic kind, the general conclusions of this study are based also on the changes in work capacity and blood pressure, and these findings fully support the subjective data.

Effects of Cordite Products.—Nitroglycerine and nitrocellulose are the main constituents of cordite, with a smaller amount of carbamite. Nitrocellulose is not volatile; carbamite has a very low vapour pressure; nitroglycerine has also a low vapour pressure, but has an exceptionally powerful pharmacological effect.

The gaseous products found after "ageing" of cordite are nitrous fumes, carbon dioxide, carbon monoxide, and an unidentified acrid component. The presence of nitroglycerine vapour was inferred from vapour-pressure considerations.

The circulatory effects (headache and fall in blood pressure) experienced by our subjects during cordite trials resembled closely those known to occur after the ingestion or inhalation of nitroglycerine, both in treatment and during the manufacture of explosives (Rabinowitch, 1944). Apart from nitroglycerine, other possible causes of the headache are the high carbon dioxide concentration and carbon monoxide poisoning.

Carbon dioxide was not of itself causative since, although it was present in comparable amounts in all trials, no headaches occurred on dummy days, either during exposure or on entering the fresh air. The latter, or "paradox" effect, was not produced, presumably because carbon dioxide at 3 to 4 per cent. was breathed for a relatively short time. Furthermore, headaches often came on in the first hour of the cordite trial before carbon dioxide had reached 1 per cent. By increasing pulmonary ventilation, carbon dioxide would, presumably, increase the absorption of nitroglycerine vapour (and other gases present).

Carbon monoxide was present in such small concentrations that it is unlikely to have had any effect, and was present on one "dummy" trial (May 9, 1946) in amounts comparable to one of the "headache-producing" cordite trials (May 31, 1946). The maximum figure for any trial was 0·002 per cent. (average for trial, 0·0017 per cent.) on May 7, 1946. This is quite unlikely to have caused symptoms. The dangerous limit for continuous work is given as 0·01 per cent. by the Air Ministry. Killick (1936) found negligible symptoms from continuous exposure of over four hours to 0·04 per cent. carbon monoxide while resting. It will be recalled however, that nothing is known about the maximum amounts of carbon monoxide which may occur inside cylinders of "aged" cordite, and the possibility therefore that toxic concentrations of carbon monoxide (and also nitrogen dioxide) may be produced from a batch of stoved cordite cannot be ruled out.

As the present experiments show, heat alone can cause a reduction in the blood pressure of working subjects. Moreover, the collapse noted in association with low systolic blood pressure is similar to heat syncope (Weiner, 1938). Comparison of hot dummy and cordite trials, however, shows that cordite was specifically responsible for the adverse circulatory effects noted in the present trials.

Nitroglycerine, therefore, remains as the likely factor producing the ill-effects observed in the present experiments. The presence of nitroglycerine vapour could not be proved chemically, but, as already stated, it was calculated to be present in minute quantities. The vapour of nitroglycerine is known to be a powerful poison even when much diluted with air (Laws, 1898), and it must be assumed that these minute quantities were absorbed through the lungs in amounts sufficient to produce adverse effects in our non-immune men.

TABLE 8

CHANGE IN SYSTOLIC BLOOD PRESSURE DURING CORDITE TRIALS COMPARED WITH THAT DURING DUMMY TRIALS*

	Temperate conditions		Hot conditions	
	Sitting	Standing	Sitting	Standing
No. of observations distributed in pairs	20	20	30	30
Degrees of freedom				
Cordite vs. dummy	1	1	1	1
Experimental days	9	9	14	14
Error=N	9	9	14	14
Total	19	19	29	29
Mean difference between cordite and dummy trials in change of Sys. B.P. (m.m.) (d)	11·1	19·3	13·4	15·07
Standard deviation of				
d	4·38	7·12	3·34	2·41
t	2·53	2·71	4·02	6·24
N	9	9	14	14
P	0·05-0·02	0·05-0·02	0·001	0·001

* The mean difference between the S.B.P. changes in the two series is estimated taking results of each man separately for "protected" and "unprotected" observations. This eliminates variance due to "experimental days". The variance due to the two sets of clothing and the interaction between clothing and experimental treatment (i.e. cordite or dummy) remain within the error variance.

To determine the role played by nitroglycerine in producing these circulatory effects, further investigation is required, using a pure source of nitroglycerine such as dynamite (nitroglycerine in Kieselguhr). A study using amounts comparable to the theoretical quantities which our subjects may have absorbed is clearly indicated.

The irritant effects of cordite on the nasal mucosa are probably not due to nitrous fumes. The reasons for ruling out nitrogen dioxide are first its distinctive and recognizable odour, and, secondly, the extremely low concentration in which it was present in the air of the trial room. The maximum found (0·00067 per cent.) is below the maximum concentration of 0·001 per cent., allowable for an exposure of several hours' duration. Lehmann (1939) states that 0·006 per cent. is required to produce moderate irritation of the larynx, and that 0·004 per cent. could be tolerated for several hours by many persons.

The clinical features of nitrous fume poisoning include changes in radiographic appearances of the lungs (Camiel and Berkhan, 1944). Methæmoglobin formation, and other alterations in the blood picture have been reported—relative lymphocytosis, eosinophilia, delayed sedimentation rate (Pfenninger, 1944), and an increase in blood platelet count.

None of the examinations made on these points provides evidence of toxic nitrogen dioxide effects. Radiographs of chests showed no abnormality; methæmoglobin could not be detected in any but insignificant amounts (one estimation only); Dr. Case's blood cytological studies also failed to show abnormalities. It is unlikely that the present methods for estimating nitrite in body fluids would reveal the small amounts absorbed either as nitrous fumes or from inhaled nitroglycerine.

Mode of Absorption.—Apart from ingestion by mouth, it is generally recognized that nitroglycerine may be absorbed both by inhalation and through the skin (Rabinowitch, 1944). Earlier investigators believed that during the cordite handling inhalation of the fumes alone could induce the symptoms. The use of protective clothing in the present trials confirms this belief, since all the adverse effects could be produced in subjects wearing the protective garments. In subjects not so protected there is no doubt that skin absorption of nitroglycerine was possible. The content of nitroglycerine in the anti-flash gloves after cordite handling was appreciable (see under "Results").

Susceptibility and Immunity.—There is a wide variation in susceptibility to nitroglycerine (Rabinowitch, 1944). One of our eight subjects appeared to be unaffected in any way throughout the trials; on the other hand the authors suffered headaches every time they were exposed.

All but 2 to 3 per cent. of nitroglycerine workers acquire an immunity to nitroglycerine after three or four days (Ebright, 1914) which may, however, be lost over the week-end (presumably two days). The workers are said to rub nitroglycerine into their hatbands to avoid "Monday headache." This immunity is said to be lessened by hot weather and by indulgence in alcohol.

Our programme was planned to allow three days between cordite trials for the subjects, so as to reduce or eliminate any immunity from the previous exposure. It was, however, difficult to avoid slight exposure on intervening days due to diffusion from the closed cordite cases during the laborious procedure of changing the cases at the end of a trial in preparation for the next. This fact would help to explain the lessened evidence of ill-effects on our second cordite exposures. It seems likely also that a pause of three days is not entirely sufficient to abolish acquired immunity.

Summary

The effects were determined of "aged" cordite on the efficiency of young adults handling it in the form of 96 lb. quarter charges, at a moderate rate of work in a small airtight compartment.

At ordinary room temperature cordite products produced no definite impairment of the ability to perform this type of work.

At wet-bulb temperatures of the order of 86° F. the efficiency of the men was definitely reduced.

The main symptoms were those of nitroglycerine poisoning. Nitrous fumes and carbon monoxide were liberated when the cordite cases were opened, but in quantities unlikely to cause symptoms.

Comparative trials conducted on a group of men protected against the absorption of cordite through the skin by impermeable rubber gloves and aprons, suggested that the main channel of absorption of cordite products was by inhalation.

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REFERENCES

Camiel, M. R., and Berkhan, H. S. (1944). *Radiology*, **42**, 175.
Ebright, G. E. (1914). *J. Amer. med. Ass.*, **62**, 201.
Killick, E. M. (1936). *J. Physiol.*, **87**, 41.
Laws, G. C. (1898). *J. Amer. med. Ass.*, **31**, 793.
Lehmann, K. (1939). Quoted in M.R.C. Special Report. "Chronic Pulmonary Disease in South Wales Coal Miners." Part II, Environmental Studies (1943).
Pfenninger, E. (1944). *Bull. Hyg. Lond.*, **19**, 363.
Rabinowitch, I. M. (1944). *Canad. med. Ass. J.*, **50**, 199.
Weiner, J. S. (1938). *J. industr. Hyg.*, **20**, 389.

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THE RISK OF FLUOROSIS IN MAGNESIUM FOUNDRIES

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Introduction

Magnesium alloys were first used in foundries in about 1935, but since then, and particularly with the expansion of the aircraft industry during the war 1939–45, their use has been extensive. According to Ford and Stern (1944), 530,000,000 pounds were used in the United States in 1944 in contrast to 6,000,000 pounds in 1939. The greatest risk in magnesium foundries is fire. Serious fires occurred in Germany in 1939; in Little Falls, New Jersey, in June, 1939; at Hounslow, England, in February, 1940; in Cleveland, Ohio, in December, 1940; and in New York in January, 1944. The alloys are safe except when the metal is molten or in finely divided form. When the metal is melted it rapidly oxidizes and ignites unless (*a*) air is excluded from the surface by a flux which usually contains fluoride, or (*b*) air is replaced by a reducing gas such as sulphur dioxide. Special oxidation inhibitors such as boric acid, sulphur, borax, or soluble fluorides, when added to moulding sands make it possible to pour molten magnesium into damp sand moulds without the danger of fire or explosion. It is evident, however, that if fluorides are used as a flux for the metal, and in the moulding and core sands, workers in such foundries might be exposed to a risk of fluorosis from atmospheric contamination.

Previous Investigation

To investigate this risk Largent and Ferneau (1944) collected spot samples of urine from men engaged in magnesium founding and analysed them for fluorine. They showed by controlled experiments that the fluorine concentrations in spot urine samples differed somewhat from the concentration in a twenty-four-hour urine sample from the same individual but the mean concentration for an entire group of spot samples did not differ significantly from that of a group of twenty-four-hour samples. This work was carried out in three foundries, and the results are shown in Table 1.

TABLE 1

MEAN FLUORINE CONCENTRATIONS IN P.P.M. IN SPOT SAMPLES OF URINE COLLECTED FROM THREE MAGNESIUM FOUNDRIES

(from Largent and Ferneau 1944)

Foundry	No. of Samples	Mean and its P.E.*	S.D.†	C. of V.‡
A	123	2·23±0·087	1·4317	64·16
B	12	3·13±0·381	1·9592	62·70
C	6	6·39	5·5695	86·87

* Probable error of the mean; † standard deviation; ‡ coefficient of variation.

In foundry A the numbers were sufficiently large to analyse the results according to the part of the foundry in which the men were working. The results are given in Table 2.

Table 2

MEAN FLUORINE CONCENTRATIONS IN P.P.M. IN URINE SAMPLES OBTAINED FROM WORKMEN IN FOUNDRY A; GROUPED ACCORDING TO PLACE OF WORK IN FOUNDRY

(from Largent and Ferneau, 1944)

Place of Work	Mean and its P.E.	S.D.	C. of V.
Core Spray ..	3·45±0·388	2·821	81·56
Pouring	2·13±0·157	1·250	58·71
Knock-out ..	1·97±0·105	1·031	52·44
Melting	1·56±0·118	1·175	75·28
Core room ..	1·50±0·087	0·997	66·45
Fettling	1·37±0·142	1·015	74·26

Two air samples from foundry B, both collected in the knock-out area, showed fluorine concentrations of 31 mg. and 84 mg. per 10 cubic metres.

The concentration was 60 mg. per 10 cubic metres in one air sample collected in foundry A in the core-setting area between the pouring and knock-out areas. It was assumed that each worker breathed 10 cubic metres of air per day. The authors considered there was evidence that workers in these occupations were exposed to a risk of developing fluorosis. In view of these results it was decided to investigate conditions in a magnesium foundry in England.

Present Investigation

The foundry investigated, in a country town in Southern England, was producing castings of magnesium-aluminium-zinc, magnesium-aluminium-manganese, and magnesium-aluminium- manganese-silver alloys. The following processes were carried out: the metal was melted in open crucibles or in an almost enclosed bale-out type of furnace; the flux used in every case contained a considerable amount of fluoride (about 15 per cent.); before pouring, the crust which formed on the melt was removed and a fluoride-containing dusting powder was sprinkled on top of the molten metal. Aluminium fluoride, to the extent of about 2 per cent. of the final mix, was added, either as solid or solution, to the sand used for moulding. Thus, possible sources of danger from fluoride were as follows: (1) in adding the flux to the open crucibles; (2) in the application of dusting powder to the molten metal; (3) in sand preparation; (4) in casting and in the process of knocking out.

The moulds were filled with sulphur dioxide from a cylinder immediately before pouring, a considerable amount being given off into the atmosphere, particularly during pouring. The high concentration of sulphur dioxide in the air was partly due to the fact that the dusting powder contained 60 per cent. sulphur.

Fluorine Content of Dust Samples.—Dust samples were collected on a salicylic acid filter. The volume of air sampled was recorded and the mass concentration of dust at the various sampling points calculated from the weight of dust obtained after dissolving out the salicylic acid in ethyl alcohol. The alcohol washings were retained and analysed for fluorine and this figure was combined with that for the fluorine found in the dust. The determination of fluorine was carried out using a modification of the method of Willard and Winter (1933). No attempt was made to trap gaseous fluorine, but it is possible that some of this might have been retained by the salicylic acid filter bed.

Thermal precipitator samples were taken near the open crucibles; the slides showed a cloud of fine, white, very hygroscopic particles, presumably from flux or dusting powder.

Population of Workmen Examined.—About 190 were employed in the foundry and core shop, and 124 of these volunteered to be examined. It was not possible to examine all the workers, or even a randomly selected cross-section of them, owing to (*a*) staggered holidays, (*b*) the progressive reduction in personnel on the grounds of redundancy while the investigation was in progress, and (*c*) the reluctance of some men to participate. The volunteers included, however, workers in the different occupations, with varying durations of exposure and of varying ages, as is shown in Tables 4 and 5. On the other hand, as stated below, only some of these men submitted to a complete examination, with radiograph and with blood and urine tests. While it is impossible to maintain with certainty that these varying samples of the men are representative of all those involved, it is believed that they are sufficient to supply some of the necessary data and give at least a broad indication of the possible risks of fluorosis in this environment.

Nature of Examination.—The examination included occupational and clinical histories, physical examination, chest expansion measurements, and vital capacity.

On the first two days of the enquiry a sample of 10 ml. of venous blood was collected from alternate men examined, but when it became evident that the total

TABLE 3

MASS CONCENTRATION AND FLUORINE CONTENT OF AIRBORNE DUST SAMPLES TAKEN AT VARIOUS POINTS IN THE FACTORY

Sampling point	Vol. of sample (m^3)	Dust Mg./m^3	Fluorine Mg./m^3
Core shop	10·2	3·3	0·143
Foundry proper:			
Mixing mill	10·2	12·5	6·370
Machine mould	10·2	3·5	0·286
Near mechanical feed	10·2	8·3	0·714
Near furnaces 1	10·2	4·3	0·314
„ „ 2	Not recorded	—	Fluorine in dust 7·5%

TABLE 4

DISTRIBUTION OF WORKERS EXAMINED BY AGE AND OCCUPATION

Occupation	No.	Age in years			
		15–29	30–39	40–49	50–59
Moulders	45	10	24	9	2
Machine moulders	8	3	4	1	—
Furnacemen	16	3	8	4	1
Crane drivers	3	—	2	1	—
Labourers	23	6	11	3	3
Core makers	26	10	10	4	2
Core oven attendants	3	—	1	1	1
Total	124	32	60	23	9

TABLE 5

DISTRIBUTION OF WORKERS EXAMINED BY YEARS OF WORK IN MAGNESIUM FOUNDRIES

Occupation	No.	Years of work		
		Under 2	2–5	6 and over
Moulders	45	14	13	18
Machine moulders ..	8	—	4	4
Furnacemen ..	16	1	7	8
Crane drivers ..	3	2	—	1
Labourers	23	8	14	1
Core makers	26	2	9	15
Core oven attendants	3	2	1	—
Total	124	29	48	47

number of volunteers would be smaller than anticipated a sample was collected from every man on the two subsequent days. From this a full blood count was taken and the rest was stored for fluorine determination. Of the 80 samples of blood thus obtained, 17 were taken into waxed tubes in order to prevent absorption of fluorine by the glass, but the remaining 63 were in unwaxed tubes. The results were expressed as parts per million whole blood. Although the mean figure for the waxed tubes was higher than that for the unwaxed the difference is not large and not more than might easily be due to chance (Table 6). In subsequent analysis the two sets of figures have, therefore, been combined.

TABLE 6

Method of storage	No.	Mean F.p.p.m.
Unwaxed tubes	63	2·48
Waxed tubes	17	2·84
Difference		0·36
Statistical significance—	"t"—0·9021. P = ·3 to ·4	

The men were asked to co-operate by collecting twenty-four hour specimens of urine at the week-end in order to avoid contamination in the foundry, but only nine were willing to do this. It was decided, therefore, to obtain as many spot samples as possible; these were given by 57 workers. To avoid contamination these specimens were passed in the ambulance room under medical supervision, and were collected in waxed bottles. The fluorine content of these samples was expressed as parts per million and the specific gravity of each specimen was recorded.

Radiographs were taken of the chest, jaw, spine, pelvis, and radius and ulna. The chest radiographs were taken with a different mobile *x*-ray apparatus from that used for the bone radiographs; they were, therefore, not done on the same day, and accordingly the numbers do not correspond exactly, since some of the men did not attend on both occasions.

Findings

Incidence of Reticulation.—Radiographs of the workers' chests showed evidence of reticulation. This was of the type found in foundries in general; it has been the subject of another investigation, which will be published later. The exact incidence, arranged according to the age of the workers and the length of exposure in foundries, is recorded in Tables 7 and 8. Seventy-eight of the men had their chests radiographed.

TABLE 7

INCIDENCE OF RETICULATION BY AGE AND OCCUPATION

Occupation	Total	Age in years			
		15–29	30–39	40–49	50–59
Moulders ..	13 (28)	3 (7)	6 (15)	2 (4)	2 (2)
Machine moulders	1 (3)	1 (1)	— (2)	—	—
Furnacemen	3 (11)	— (2)	1 (6)	1 (2)	1 (1)
Crane drivers	— (2)	—	— (2)	—	—
Labourers ..	5 (16)	1 (5)	1 (6)	1 (3)	2 (2)
Core makers	5 (15)	— (4)	3 (6)	1 (3)	1 (2)
Core oven attendants	1 (3)	— (1)	— (1)	—	1 (1)
Total ..	28 (78)	5 (20)	11 (38)	5 (12)	7 (8)

(Figures in brackets show the total number radiographed)

TABLE 8

INCIDENCE OF RETICULATION BY YEARS OF WORK IN MAGNESIUM FOUNDRIES

Occupation	Total	Years of work		
		Under 2	2–6	Over 6
Moulders	13 (28)	3 (9)	5 (13)	5 (6)
Machine moulders ..	1 (3)	—	1 (2)	— (1)
Furnacemen	3 (11)	— (1)	2 (5)	1 (5)
Crane drivers ..	— (2)	— (2)	—	—
Labourers	5 (16)	— (8)	5 (5)	— (3)
Core makers	5 (15)	— (1)	2 (5)	3 (9)
Core oven attendants	1 (3)	1 (1)	— (1)	— (1)
Total	28 (78)	4 (22)	15 (31)	9 (25)

Vital Capacities.—The vital capacities of the workers (Table 9) showed no significant difference between the groups, except that the latest recruits to the foundry had a rather low vital capacity. The reason for this is not evident, but it might be due to the low standard of physical fitness in those rejected for military service and thus left available for work in industry. Since the radiographs were taken before the clinical examinations were made, the numbers are not identical for the two investigations. Some men were unwilling to attend at the second visit, while some had been

discharged on grounds of redundancy since the factory was closing.

Fluorine.—The fluorine content of blood and urine samples was determined by a modification of the method of Willard and Winter (1933); the results are given in Table 10.

The nine subjects who gave twenty-four hour specimens of urine were found to be excreting from 0·35 (0·4 p.p.m.) to 3·80 (2·8 p.p.m.) with a mean of 1·10 mg. fluorine every twenty-four hours (1·3 p.p.m.). A summary of the excretion of fluorine in the 53 spot samples, grouped according to years of exposure, is given in Table 13. The specific gravities of these samples were in the range of 1·005 to 1·037, with a mean of 1·027. These results show, by comparison with the control series, that there is a slightly increased excretion of fluorine in all the groups of workers except those who had been at the foundry for less than two years.

In the groups labelled "core shop" and "furnacemen" there was no significant difference between those who had been exposed for more than 6 years and those with shorter exposure. The difference between the three exposure groups amongst the foundry workers is more than is likely to be due to chance, and shows a progressive increase in fluorine excretion with longer exposure. The mean value for the urinary fluorine excretion of all the workers examined in this magnesium foundry is 2·50 p.p.m., which is very close to the mean value of 2·48 observed by Largent and Ferneau for 141 subjects (Table 1).

It is unlikely that the increased excretion of fluorine was due to a high intake in the drinking water, because the fluorine content of the water supply in the district in which the foundry was situated, and of that in which the workers lived, was 0·15 and 0·18 p.p.m. respectively, whilst the water supply used by the control group from a town in England, some miles distant from the foundry, contained 0·09 p.p.m., and the two supplies available to the other control group in Scotland contained 0·07 and 0·18 p.p.m.

On the other hand the blood fluorine level in the different groups of workers at the foundry (Table 11) showed no significant difference with duration of exposure. These figures, giving a mean of 2·58 p.p.m. can be compared with a group of residents in Scotland, and reveal no significant difference.

Blood Counts.—The results of the hæmatological examination are given in Table 12. The hæmoglobin was estimated by the photoelectric alkaline hæmatin method. (13·8 mg. per 100 g.=100 per cent.) These results show no abnormalities.

Radiographic Changes.—Radiographic changes rarely become manifest until the worker has been exposed to excessive quantities of fluorine for more than seven years (M.R.C. Report on fluorosis in Fort William—to be published). Since magnesium founding has only been performed for some ten years, and only extensively for about five years, the number of men exposed for a sufficient length of time to show these changes must necessarily be few. Only one man in this foundry showed radiographic changes in his bones suggestive of fluorosis. No changes which could be considered significant were seen in other men who had their bones radiographed.

Case Report

A man aged 37 had seen service in the army and four years in agricultural work before coming to the magnesium foundry to work as a furnaceman on the open crucible furnaces, which he had done for the past five and a half years. He had no symptoms and no abnormal physical signs. He had had three teeth extracted; two were carious and six were filled. His blood count showed 5,500,000 red cells, and 11,000 white cells, of which 61 per cent. were polymorphs, 34 per cent. lymphocytes, 2 per cent. monocytes, 2 per cent. eosinophils, and 1 per cent. basophils. The hæmoglobin

TABLE 9

VITAL CAPACITIES IN ML.

Occupation	Years in Foundry								
	Under 6						6 and over		
	No.	Mean	S.D.				No.	Mean	S.D.
Core shop	10	2,046	704				14	3,096	801
Furnacemen	6	3,292	338				9	3,006	1,011
	Under 2			2–5					
	No.	Mean	S.D.	No.	Mean	S.D.			
Foundry	20	2,775	638	22	3,057	631	22	3,264	754

TABLE 10

FLUORINE CONTENT OF BLOOD AND URINE SAMPLES

No.	Age	Fluorine Content p.p.m.			Urine 24-hr. samples mg. F. per 24 hrs.	No.	Age	Fluorine Content p.p.m.			Urine 24-hr. samples mg. F. per 24 hrs.
		Blood	Urine					Blood	Urine		
			Spot Samples						Spot Samples		
			F.p.p.m.	Sp. Gr.					F.p.p.m.	Sp. Gr.	
			Core Shop			49	33	—	1·0	1·016	—
1	48	—	3·8	1·024	—	50	30	2·4	1·4	1·028	—
2	39	1·4	3·5	1·031	—	51	42	1·5	—	—	—
3	55	—	1·7	1·037	—	52	41	1·8	—	—	—
4	26	0·8	2·8	1·025	0·78	53	45	2·4	—	—	—
5	23	4·2	0·9	1·031	—	54	42	2·5	—	—	—
6	23	1·5	3·3	1·031	—	55	35	2·5	3·7	1·029	—
7	21	2·0	—	—	—	56	37	2·1	—	—	—
8	41	3·0	—	—	—	57	30	1·5	2·3	1·026	—
9	34	4·7	—	—	—	58	29	2·9	—	—	—
10	37	1·6	3·0	1·026	—	59	37	1·5	—	—	—
11	32	4·7	0·9	—	—	60	45	1·9	5·3	1·027	—
12	34	2·6	1·6	1·028	—	61	33	1·7	—	—	—
13	44	—	2·8	1·029	—	62	32	—	2·6	1·024	—
14	25	4·3	4·0	1·024	—	63	26	—	0·9	1·029	—
15	22	4·2	1·4	1·029	—	64	22	8·4	—	—	—
16	36	1·2	—	—	—	65	52	—	3·8	1·036	—
17	30	1·1	—	—	—	66	31	2·6	—	—	—
18	36	2·0	1·9	1·022	—	67	37	4·5	—	—	—
19	47	3·9	—	—	—	68	42	4·2	5·1	1·026	—
20	24	1·3	4·1	1·036	—	69	34	6·1	7·5	1·028	—
21	20	1·5	2·9	—	—	70	34	3·1	1·5	1·025	—
22	54	2·3	—	—	—	71	31	1·5	—	—	—
23	53	2·3	1·9	—	—	72	32	2·6	2·0	1·035	—
24	39	2·2	—	—	0·35	73	37	1·5	—	—	—
25	24	5·3	1·7	1·020	3·8	74	46	1·7	—	—	—
						75	40	—	1·2	1·030	—
			Foundry			76	30	0·9	—	—	—
26	29	3·9	2·7	1·031	—	77	22	—	2·5	1·030	—
27	31	2·6	—	—	—	78	22	2·9	—	—	—
28	25	1·9	—	—	—	79	22	1·9	—	—	—
29	42	—	2·7	1·026	—	80	28	—	1·8	1·036	—
30	38	2·9	3·7	1·026	—	81	36	—	—	—	0·74
31	33	1·6	2·6	1·024	—	82	38	1·6	—	—	0·78
32	31	1·1	0·5	1·031	—	83	31	1·8	1·4	—	1·10
33	33	2·0	2·6	1·028	—				Furnace-men		
34	33	1·0	—	—	—						
35	22	2·6	3·0	1·032	—	84	45	3·6	2·2	1·027	—
36	29	2·6	1·0	1·027	—	85	28	0·6	—	—	—
37	23	2·3	—	—	—	86	32	2·1	—	—	—
38	25	—	4·5	1·037	—	87	30	1·4	1·5	1·033	—
39	39	—	1·1	1·029	—	88	36	—	2·7	1·024	—
40	38	1·9	6·9	1·029	—	89	29	1·7	—	—	—
41	35	1·4	1·0	1·026	—	90	36	2·3	—	—	—
42	49	2·0	—	—	—	91	46	5·0	—	—	—
43	40	2·3	1·8	1·028	—	92	50	4·7	—	—	—
44	57	1·2	—	—	—	93	30	3·5	—	—	—
45	34	7·1	—	—	—	94	37	4·6	—	—	—
46	31	—	1·6	1·026	—	95	32	1·0	1·0	1·005	0·98
47	34	1·5	—	—	—	96	33	1·6	2·6	1·017	0·39
48	38	—	2·3	1·023	—	97	46	2·5	3·4	1·021	1·10

TABLE 11

BLOOD FLUORINE IN P.P.M. BY LENGTH AND TYPE OF EXPOSURE

Occupation	Years in Foundry								
	Under 6						6 and over		
	No.	Mean	S.D.	No.	Mean	S.D.	No.	Mean	S.D.
Core shop	8	3·41	1·14				14	2·34	1·00
Furnacemen	5	3·02	1·52				8	2·44	0·80
	Under 2				2–5				
Foundry	10	2·57	1·65	15	2·61	1·72	20	2·35	1·19
	Control Series								
	No.	Mean	S.D.						
Scotland	20	2·77	1·23						

TABLE 12

DETAILS OF BLOOD COUNTS

	Years in Foundry								
	Under 6 years (9)						6 and over (14)		
Core shop	%	Mean	S.D.				%	Mean	S.D.
Hæmoglobin		109	4·45					106	8·0
Red cells		5·54	0·26					5·28	0·43
White cells		8·12	1·07					9·00	1·74
Polymorphs	60		10				60		7
Lymphocytes	36		7				35		3
Marocytes	4		2				4		2
Eosinophils	1		1				1		1
Basophils	0		0				0		0
Furnacemen			(5)						(9)
Hæmoglobin		107	12·15					109	6·96
Red cells		5·57	0·23					5·27	0·24
White cells		9·60	0·83					8·47	2·22
Polymorphs	61		2				61		5
Lymphocytes	31		3				35		7
Marocytes	5		1				4		2
Eosinophils	2		1				1		1
Basophils	1		—				0		0
	Under 2 (14)			2–5 (18)			6 and over (21)		
Foundrymen	%	Mean	S.D.	%	Mean	S.D.	%	Mean	S.D.
Hæmoglobin		113	8·21		110	5·62		110	7·06
Red cells		5·53	0·43		5·53	1·32		5·44	0·12
White cells		7·05	1·22		7·78	1·98		7·11	2·05
Polymorphs	61		4	62		7	63		4
Lymphocytes	33		4	32		6	31		4
Marocytes	5		2	5		2	5		2
Eosinophils	1		1	1		1	2		2
Basophils	0		0	0		0	0		0

Number of men in brackets

TABLE 13

FLUORINE IN SPOT SAMPLES OF URINE IN P.P.M. GROUPED ACCORDING TO LENGTH AND DEGREE OF EXPOSURE

Occupation	Years in Foundry								
	Under 6						6 and over		
	No.	Mean	S.D.				No.	Mean	S.D.
Core shop	8	2·40	1·09				9	2·56	0·96
Furnacemen	2	2·10	0·60				4	1·98	0·59
	Under 2			2–5					
	No.	Mean	S.D.	No.	Mean	S.D.			
Foundry	9	1·60	0·49	10	2·78	1·64	11	3·92	1·93
	Control Series								
	No.	Mean	S.D.						
England	20	1·58	0·74						
Scotland	20	0·51	0·29						

was 112 per cent. His blood contained 4·6 p.p.m. fluorine. The urine was not examined. The radiographs showed a uniform increase of density of the bones, ossification of the ligamentous attachments, and increased density of the tips of the spinal processes (figs. 1 and 2, p. 231).

Conclusion

It may be suggested, in view of the average raised level of fluorine excretion in the urine of the workers and the fact that one man showed radiological evidence of bone changes, that at least some of the workers in the atmosphere of this magnesium foundry were exposed to a risk of fluorosis.

Roholm (1936) established 15 to 25 mg. per day as the probable toxic dose for cryolite. Based on this, Williams (1942) has suggested 3 p.p.m. (2 mg. per cubic metre) as a tentative upper limit for fluorides in magnesium foundries. The concentrations of particulate fluorine in the foundry investigated in Great Britain were only between 0·143 and 0·714 mg. per cubic metre, except for a sample taken near the mixing mill, which showed a concentration of 6·37 mg. per cubic metre. There is, however, no conclusive evidence that 3 p.p.m. is too high a concentration of atmospheric fluoride for safety, because conditions in this foundry are different in different sections and a man would not be exposed to such a concentration throughout his eight-hour period of work. Nevertheless, the results of this enquiry suggest that improved methods of production should aim at reducing atmospheric contamination by fluorine compounds if such a magnesium foundry is to be entirely free from risk.

Summary

The results of an investigation into the possible occurrence of fluorosis in workers in a magnesium foundry are described.

Out of 124 persons examined, radiological examination showed only one case suggestive of fluorosis, but only 47 of the workers had been exposed to risk for as long as six years.

There was an increased excretion of fluorine in the urine of the workers in the foundry when compared with two control groups. There was no excess of fluorine in the blood.

The fluorine content of dust collected from the air in the foundry varied between 0·143 and 6·37 mg. fluorine per cubic metre. The possible effect of these conditions on the health of the workers is discussed.

We would like to express our gratitude to Dr. A. G. Eddison and the management of High Duty Alloys, Ltd., for their co-operation. We are also indebted to Messrs. H. T. Ferrier and J. Maclagan for taking the *x* rays; and to Dr. M. H. Jupe for his help in their interpretation.

REFERENCES

Ford, C. B., and Stern, A. C. (1944). *Industr. Hyg. Bull.*, **23**, 253.
Largent, E. J., and Ferneau, I. F. (1944). *J. Industr. Hyg.* **26**, 113.
Roholm, K. (1936). *Klin. Wschr.* 15, 1425.
Willard, H. H., and Winter, O. B. (1933). *Ind. Eng. Chem.* (Anal. Edit.), 5, 7.
Williams, C. R. (1942). *J. Industr. Hyg.* **24**, 277.

For Illustrations of this Article, see page 231

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THE EXPERIMENTAL PRODUCTION OF X-RAY SHADOWS IN THE LUNGS BY INHALATION OF INDUSTRIAL DUSTS: I. IRON OXIDE

BY

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Our endeavour is to obtain a better understanding of the cause of radiographic shadows following the inhalation of industrial dusts, and to correlate the radiological and histological findings. This paper describes attempts to produce similar changes in the lungs of animals from inhalation of one such dust.

Harding (1945) showed that iron oxide, suspended in saline and injected into the tracheas of rats, results in radiographic shadows in the lungs similar to those occurring in siderosis in man, and that these shadows are due solely to iron oxide. Such a technique is highly artificial and bears very little relation to the inhalation of dust into the lungs under industrial conditions: it is even conceivable that the reactions in the lungs might be different under the two circumstances. In the present series of experiments an attempt is being made to reproduce as nearly as possible the atmospheric conditions occurring in industry.

Material

Our intention was to use "rouge," which is nearly pure Fe_2O_3, but when this material is stirred electrostatic phenomena result in its aggregation into rock-like lumps so that it is impossible to produce a cloud of dust. We were therefore driven to use "crocus," a less pure form of iron oxide. Both of these dusts are, however, used by silver polishers (McLaughlin and others, 1945). Analysis of the crocus used showed its percentage composition to be Fe_2O_3, 81·5; Na, Ca, Mg, K, all above 1 and below 3, As 0·012, Pb 0·01, Cu 0·003, Mn 0·003, Ni 0·01, total silica 0·6, free silica 0·2.

Method

Twelve rats, 2 months old, average weight 128 g., were exposed to inhalation of dust in the animal chamber and dust feed apparatus previously described (Lloyd Davies, 1946). Exposure was continued for 3 hours daily for 140 days: the average dust count in the chamber was maintained at 1,500 to 2,000 particles per ml. for 16 days, then increased to 2,500 to 3,000 for 47 days, and finally increased to 5,000 particles per ml. for 77 days. The differential count showed that 88 per cent. of the particles were 1 μ or less in diameter.

Two animals appeared ill during the experiment and were killed; subsequently animals were sacrificed at intervals from 80 days to 235 days following cessation of exposure. The animals were anæsthetized with ether, the trachea exposed and ligated, and the animals killed by cutting the abdominal aorta (through an incision previously made in the abdominal wall). The lungs were subsequently removed in an inflated condition, and preserved in formol saline. After being radiographed, the lungs were examined histologically. Portions of several lobes were fixed for a further period in a saturated aqueous solution of corrosive sublimate, and sections were stained with hæmatoxylin and eosin, Weigert's elastin stain and van Gieson, and by Wilder's modification of Foot's method for reticulin fibrils: a few sections were also stained with potassium ferrocyanide and HCl, and a few were examined after incineration at 500° C. There were no essential differences in the lungs of animals killed at the varying intervals after exposure, and, in order to avoid repetition and because they afforded the longest opportunity for the development of a permanent tissue reaction, only the lungs of the three animals killed 235 days after the period of exposure are described here. No animals were kept for longer periods because of the risk of development of "spontaneous" bronchiectasis.

Morbid Anatomy

There was a fairly considerable variation in the amount of dust in the lungs of different animals sacrificed at the same period. There is no reliable method of measuring the degree of this difference, which we have noticed also with other dusts, but it may well be of the order of 100 per cent. Because of this animal variation, which is a marked but

insufficiently recognized feature in human pathology, and because of the small number of animals involved, we do not place much weight on the impression that the quantity of dust present became steadily less as the period after exposure lengthened. None of the animals showed as great a concentration of dust in the lungs as is found in the organs of many industrial workers, and we are taking steps to increase the total quantity inhaled in future experiments. Nevertheless, a sufficient quantity was present to be clearly visible to the naked eye and to show the nature of the response in the lungs.

In the lungs of the 3 animals sacrificed after the longest period, iron oxide was clearly visible as tiny red spots that appeared to be mainly in the pleura but were also clearly visible within the lung substance. The hilar lymph glands appeared free from iron oxide. Sections showed that the pigment was contained within phagocytic cells that occurred mainly in aggregates at lymph junctions. Some of these aggregates were in the pleura, but the majority were related to small blood vessels a short distance below the surface of the lung (fig. 1, p. 232). Very few cells containing pigment were found in alveolar walls or alveolar spaces, and the lymphoid tissue around the bronchi contained only occasional cells of this type. Nowhere within the lung was there any evidence of reaction to the presence of these dust-filled phagocytes (figs. 2 and 3, p. 232). The number and thickness of reticulin fibrils appeared equal in the areas of lymph junction which contained many or no phagocytes : there was no trace of collagen fibre production, and elastic fibrils were unaltered.

Radiography

The radiographs (fig. 4, p. 232) showed pin-point dense shadows located at the periphery of the lung field and corresponding with small collections of iron oxide either in the pleura or just beneath the surface of the lung. No intensification of the lung markings was apparent.

Discussion and Summary

Definite radiological shadows have been obtained from the lungs of rats after inhalation of crocus, a crude form of Fe_2O_3. These shadows correspond exactly with aggregates of phagocytes containing dust. There is no evident tissue reaction to the presence of these phagocytes and their contained dust. The radiographic shadows are produced solely by the aggregates of iron oxide.

It is a pleasure to acknowledge the cheerful work of Miss E. Wilkinson in the tedious task of attending the apparatus and recording the dust counts. The photomicrographs were taken by Mr. A. W. Collins, F.I.M.L.T.

References

Harding, H. E. (1945). *Brit. J. industr. Med.*, 2, 32.
Lloyd Davies, T. A. (1946). *Ibid.*, 3, 111.
McLaughlin, A. I. G., Grout, J. L. A., Barrie, H. J., and Harding H. E. (1945). *Lancet*, 1, 337.

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Brit. J. industr. Med., 1947, 4, 225.

ARGYRO-SIDEROSIS OF THE LUNGS IN SILVER FINISHERS

BY

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(RECEIVED FOR PUBLICATION, NOVEMBER 29, 1946)

McLaughlin and others (1945) described the clinical and radiological findings in four men who had been silver finishers for many years, and the results of an autopsy on one of them. They found that the iron oxide and silver dust to which these men were exposed in the course of their work had been inhaled into their lungs causing stippled or reticulated shadows in radiographs of the chest, but that this was not associated with obvious physical disability. In the case coming to necropsy there was emphysema but no fibrosis of the lungs; the iron pigment formed intra-cellular masses distributed throughout the course of the lymphatics, and the silver had produced intra-vitam staining of the elastica in the arterial and alveolar walls.

Three further silver finishers have since come to necropsy, and a brief description of the findings will serve to show that the changes in the lung are remarkably uniform. A summary of the original case is placed first in the list.

Iron oxide is opaque and relatively insoluble; it therefore does not give a visible blue coloration with ferrocyanide and hydrochloric acid, and in ordinary hæmatoxylin and eosin preparation it is indistinguishable from carbon and other opaque particulate matter. McLaughlin and others (1945) identified it by incinerating the sections and then examining them by reflected light. In the present study we found it better to mount the unstained sections on thin slides and examine them by dark-ground illumination. By this method the iron oxide appears bright orange-red. The silver deposits on the elastica were identified by bleaching with 1/1,000 KCN solution as described in the original paper. By reflected light the silver showed as bright glittering particles but was not thus distinguishable from other particulate matter. The results of analysis of the iron and silver content of some of the organs of these four cases are considered separately from the other necropsy findings.

Case Histories

Case 1.—A. B., aged 60, had been a silver finisher for 46 years. He had a slight cough for 16 years, and coughed up copious reddish-black sputum but made no complaint of dyspnœa. On clinical examination six years before death he was thought to have emphysema, and a radiograph of the chest showed generalized stippling or reticulation of both lung fields. Nine weeks before death he developed continuous and increasing epigastric pain from a gastric ulcer, and he died of peritonitis and bronchopneumonia shortly after a partial gastrectomy.

Necropsy Report.—Necropsy examination showed argyro-siderosis of the lungs and chronic emphysema. The patient had had a partial gastrectomy for peptic ulcer, and the gastric suture line was ruptured. The signs of general peritonitis and bronchopneumonia were present. There was bullous emphysema of the anterior borders, and generalized spongy emphysema of the rest of the lungs, but no visible fibrosis.

Microscopical Findings.—The lungs showed extensive acute bronchopneumonia and well-marked emphysema. The subpleural and perivascular connective tissue contained aggregates of phagocytes laden with opaque granules of iron oxide which appeared black in transmitted light, and in incinerated sections viewed by reflected light were seen to be reddish brown. On the elastic fibrils in the alveolar walls and the internal elastic laminæ of the smaller blood vessels was a very fine brownish black deposit of silver granules (fig. 1), which could be removed by treating the sections with 1/1,000 KCN, and which was probably silver. Iron pigment stainable with ferrocyanide and HCl was present in small amounts in intra-alveolar phagocytes.

There was moderate central lobular congestion of the liver, and in sections treated with ferrocyanide and HCl there were abundant blue granules in the liver cells and in some of the Kupffer cells. The reticulo-endothelial cells of the splenic pulp were closely packed and hypertrophied and contained masses of hæmosiderin.

Case 2.—C. W., a male aged 64 years, had been a silver finisher for 39 years and had complained of severe cough for many years. He was admitted on May 1, 1944, with intestinal obstruction due to carcinoma of the pelvic colon. A cæcostomy was performed under spinal anæsthesia and the patient had a stormy post-operative course marked by extreme tachycardia. He began to recover, but then suddenly relapsed and died on May 18, 1944.

Necropsy Report.—Necropsy examination revealed argyro-siderosis of the lungs, chronic emphysema, and carcinoma of the pelvic colon, suppurating infarct of the left lung, and hæmoptysis and hæmothorax. Laparotomy and cæcostomy had been performed. The body was of a very thin old man. There was no pigmentation of the skin, doubtful grey pigmentation of sclerotics, frothy bloody fluid throughout the trachea and bronchi, fibrinous pleurisy of both lower lobes, and 15 oz. of blood in the left pleural cavity. There was a collapsed cavity 6 cm. in diameter in the left lower lobe, communicating by a small aperture with the pleural cavity. There was an old, adherent ante-mortem thrombus in the lower branch of the left pulmonary artery, marked general emphysema, and œdema of the lungs, with bullous emphysema of the apices and anterior borders. A cut surface was uniformly dark grey. There was no enlargement of the hilar nodes. The weight of the left lung was 1 lb. 6½ oz. and of the right lung 15 oz. The small, firm liver and the kidneys showed marked chronic venous congestion. There was fusiform carcinoma of the sigmoid colon without visible secondary deposits. A cortical lipoid adenoma 1·2 cm. in diameter was present in each of the adrenals. The heart was of normal size and weighed 10½ oz. There was gross patchy calcification of the femoral arteries.

Microscopical Findings.—The lung showed severe œdema and emphysema, with an old infected infarct in the left lower lobe and abundant intracellular aggregates of iron oxide distributed fairly evenly along the course of the lymphatics. No stainable iron was found in the lungs or hilar nodes. Many of the alveolar walls were outlined by thin double lines of silver impregnation. The internal elastic lamina of most small arteries and veins was heavily impregnated with silver (fig. 2), and there was occasional focal impregnation of the external elastic laminæ, but no impregnation of either the internal or external elastic laminæ of larger arteries or veins.

Pituitary.—There were small areas of fibrosis in the anterior lobe adjacent to the pars nervosa.

Testis.—There was slight senile fibrillar thickening of the basement membranes of the tubules.

Liver.—There was moderate fat vacuolation at the periphery of the lobules, and granular bile pigment in the central cells. Sparse aggregates of stainable iron were seen in the liver cells.

Spleen.—There was cellular hyperplasia of the pulp, no stainable iron, and moderate atrophy of the Malphigian bodies.

Other Findings.—There was brown atrophy of the myocardium and muscular wall of the stomach and small gut. The colon showed well differentiated, ulcerated carcinoma with abscess formation in the wall. There were lipoid adenomata in the adrenals, and nothing of note in the pancreas, skin, aorta, or in representative sections throughout the brain.

Case 3.—S. O., a male aged 55 years, had worked as a silver finisher for 26 years. He had occasional attacks of indigestion but no habitual cough. He collapsed suddenly in a tram and died four hours later.

Necropsy Report.—Necropsy showed argyro-siderosis of the lungs, severe emphysema, hypertension, and pontine hæmorrhage. The body was of a well-built man, and there was no wasting and no pigmentation of skin or sclerotics. There were pink gelatinous thymic bodies, each lobe measuring 6 × 2 × 2 cm. The thyroid was moderately enlarged, with pinhead colloid acini. There were no pleural adhesions. The visceral pleura were mottled white and black; both lungs were bulky, with generalized emphysema, especially marked in the anterior borders and in the apices, where bullæ measured 2·5 cm. diameter. The cut surface was a uniform rusty red, and dripped with fluid. The hilar nodes were not enlarged. There was hypertrophy of the left ventricle (2 cm. thick), and slight hypertrophy of the right ventricle (0·6 cm. thick). The rather brown liver had a faint lobular pattern. The convolutions of the brain were slightly flattened. There was dark red gelatinous blood clot in fourth ventricle, maceration of ventral half of pons by recent hæmorrhage, and slight patchy atheroma of cerebral vessels.

Microscopical Findings.—The lungs (fig. 3) showed moderate œdema, severe generalized emphysema, no bronchitis, and no fibrosis. There were rather sparse intracellular iron oxide aggregates in the course of small lymphatics, but more numerous round the large arteries. There were many intra-alveolar phagocytes laden with iron oxide, but no stainable iron. The alveolar walls and internal elastic lamina of the smaller arteries and veins showed abundant linear silver impregnation, but there was no impregnation of the external elastic lamina or of the internal lamina of the larger arteries. The external elastic lamina in one big artery in the hilum was calcified. There were abundant iron oxide deposits, without fibrosis, in the hilar nodes, together with some stainable hæmosiderin.

There was considerable plasma cell, polymorph, and lymphocyte infiltration in the wall of the trachea, and a moderate amount of lymphoid tissue in the thymus.

Some thyroid acini were distended with colloid, but the majority were collapsed, with desquamated lining.

The liver and kidney showed slight arteriolar hypertrophy and there was considerable capillary congestion of the liver. Stainable iron was rare in the liver cells, and absent in the spleen and kidney. The lymphoid tissue in the spleen was atrophied. Tubules in the testis showed early focal arteriolar hyalinization and slight thickening of basement membranes. Sections of the heart, coronary arteries, carotid vessels, aorta, mucous membrane of the cheek, salivary gland, prostate, ureter, pancreas, adrenal, stomach, duodenum, and ileum, all showed nothing of note.

Case 4.—J. C., aged 65, worked as a silver finisher for 50 years and retired six months before his death. He was found dead in bed and had probably been dead for two days. He had never complained of cough or shortness of breath.

Necropsy Report.—Necropsy showed argyro-siderosis of the lungs, moderate diffuse emphysema, and hæmorrhagic pneumonia. The body was of a thin man, but there was no wasting. There was considerable muco-pus in the bronchi and lower part of the trachea, and half a pint of clear fluid in the right pleural cavity. The pleura over both lungs was bluish-black, with scattered small reddish-black plaques, especially posteriorly. There was moderate emphysema of the anterior borders of both lungs, which were markedly congested. The right lower lobe was solid, heavy, and dark red. The right ventricle of the heart was dilatated without hypertrophy. The spleen was about twice the normal size, very soft, and with prominent Malphigian bodies. There was slight venous congestion of the liver.

TABLE 1

IRON CONTENT OF DRY TISSUES IN GRAMMES OF IRON IN ONE HUNDRED GRAMMES DRY TISSUE *

	Lung	Liver	Spleen	Kidney	Heart	Aorta	Pancreas	Brain
Silver finishers : Case 1 ..	0·72	—	—	—	—	—	—	—
Case 2 ..	0·69	0·11	—	0·16	0·08	—	—	—
Case 3 ..	1·85	0·14	0·14	0·056	0·027	—	0·047	0·046
Case 4 ..	0·71	0·19	0·23	0·12	0·074	0·17	—	—
Normal : Sheldon (1935)	0·22	—	—	—	—	—	—	—
Brückmann and Zondek (1939)	—	Range, 0·07–0·157 ; mean, 0·108	—	Range, 0·029–0·054 ; mean, 0·041	—	—	—	—
Cancer Cases : (mean figures) Buchwald and Hudson (1944)	0·102	0·119	0·25	0·037	0·032	—	—	—
Coal miner with pulmonary disease Cummins and Sladden (1930)	Highest, 0·74 ; mean, 0·44	—	—	—	—	—	—	—

* Where there are no entries, no measurements were made.

TABLE 2

SILVER CONTENT OF DRY TISSUES IN GRAMMES OF SILVER IN ONE HUNDRED GRAMMES DRY TISSUE*

Silver finishers :	Lung	Liver	Spleen	Heart	Aorta	Kidney
Case 1 ..	0·06	—	—	—	—	—
Case 2 ..	0·009	0	—	0·0008	0	0
Case 3 ..	0·0013	0·0004	0·021	0	—	—
Case 4 ..	0·06	—	—	—	—	—

* Where there are no entries, no measurements were made.

Microscopical Findings.—The lungs (figs. 4 and 5) showed considerable emphysema, extreme œdema (often hæmorrhagic), but no fibrosis. There were relatively scanty iron oxide aggregates in the course of the smaller lymphatics, and more numerous ones round the large vessels, but no stainable iron. There was widespread silver impregnation of the alveolar walls, patchy impregnation of the internal elastic lamina of the smaller vessels, and considerable thickening of the intima of the pulmonary arterioles.

The heart showed brown atrophy. The splenic pulp was a mass of fused autolysed red cells with a few small granules of stainable iron. There was severe venous congestion of the kidney, with moderate atheroma of the renal vessels and slight patchy cortical ischæmia. Many of the Kupffer cells in the liver were hypertrophied and contained fine black particles. There was considerable golden-brown bile pigment in the liver cells towards the centre of the lobules, and a slight amount of stainable iron along the free borders of many of the liver columns. By dark-ground illumination the black particles in the Kupffer cells appeared shining white.

The iron and silver content of several of the organs of these cases was analysed. The silver was estimated with the use of p-dimethylamino-benzol-rhodanine (B.D.H., 1941). The results are shown in Tables 1 and 2.

Discussion

The uniformity of these four cases establishes the pattern of histological findings in the lungs after inhalation of iron oxide and silver dust. The iron oxide is inert and produces no interstitial fibrosis when taken by phagocytes into the lymphatic system. Case 3 was the only one where the man was exposed to the dust right up to the day of his death. It is significant that the iron content of his lungs was more than double that of the other three, and that his lungs were the only ones that contained great numbers of intra-alveolar phagocytes loaded with iron (fig. 3). His spleen, liver, and kidneys contained no increased iron content, and it seems likely,

therefore, that most of the inhaled iron dust is taken up by phagocytes and eliminated via the sputum. McLaughlin and others (1945) thought that the excess of stainable iron in the liver and spleen of Case 1 might have been derived from the deposits in the lungs, but in the other three cases neither the stainable iron nor the total iron content of the liver and spleen was significantly increased. The only evidence for systemic absorption of the pulmonary iron deposits was the raised iron content of the kidneys in Cases 2 and 4.

Inhaled silver dust obviously has a great affinity for the elastica of the alveolar walls and the smaller pulmonary vessels, and the large amount of the elastic tissue in the lungs probably explains why the silver does not readily gain access to skin and kidneys, which are the usual sites of deposition when argyrosis occurs from ingestion of silver.

All the cases described in this paper had considerable emphysema. The relationship of this to the silver impregnation of the alveolar walls is speculative, for in our experience most Sheffield workmen have well-marked emphysema at the age of 60. Unfortunately no radiographs were taken of these men's chests during life, but we have little doubt that they would have been similar to those described by McLaughlin and others (1945). Except that one man complained of cough for several years, there was no history of any respiratory disability in these cases.

Summary

Changes in the lungs are described in three new necropsies on silver finishers. The findings were similar to those in the one case previously recorded.

The photographs were taken by Mr. A. W. Collins and Mr. F. B. Battersby. The micro-sections were prepared by Mr. D. Bradey.

References

B.D.H. Book of Organic Reagents (1941). London.
Brückmann, G., and Zondek, S. G. (1939). *Biochem. J.*, **33**, 1845.
Buchwald, K. W., and Hudson, L. (1944). *Cancer Res.*, **4**, 645.
Cummins, S. L., and Sladden, A. F. (1930). *J. Path. Bact.*, **33**, 1095.
McLaughlin, A. I. G., Barrie, H. J., Grout, J. L. A., and Harding, H. E. (1945). *Lancet*, **1**, 337.
Sheldon, J. H. "Hæmochromatosis" (1935). Oxford University Press, London. Pp. 211.

For Illustrations of this Article, see opposite page

Legends to Illustrations

FIG. 1.—Section of lung (× 36). Silver impregnation of elastica in artery

FIG. 2.—Section of lung (× 130). Œdema and emphysema; silver impregnation of elastica of an artery

FIG. 3.—Section of lung (× 130). Intra-alveolar phagocytes laden with iron oxide; silver impregnation of the elastica of an artery and of alveolar walls

FIG. 4.—Section of lung (× 130). Emphysema and œdema; silver impregnation of elastica of artery and of alveolar wall; iron oxide limited to interstitial phagocytes

FIG. 5.—Detail from fig. 4 (× 250)

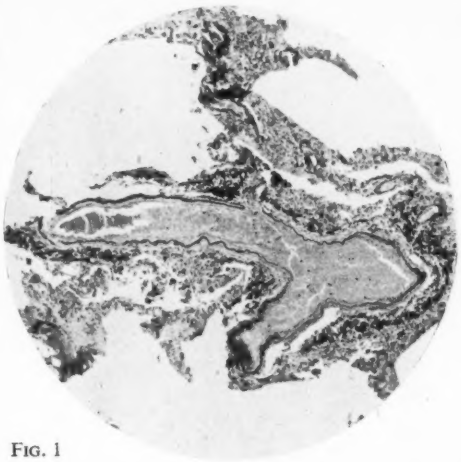


FIG. 1

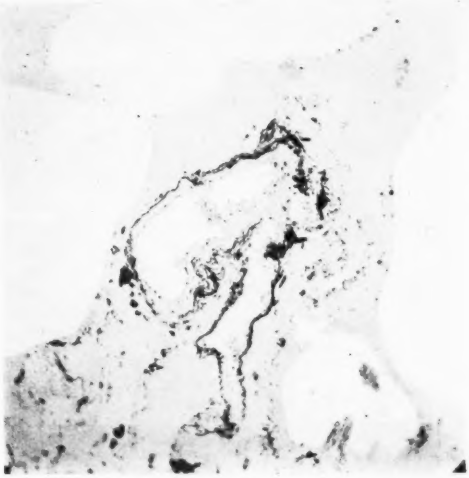


FIG. 2

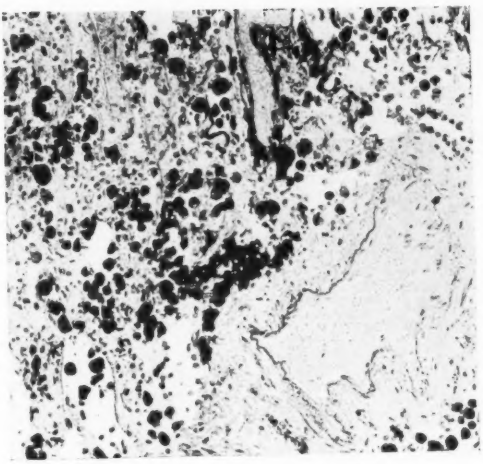
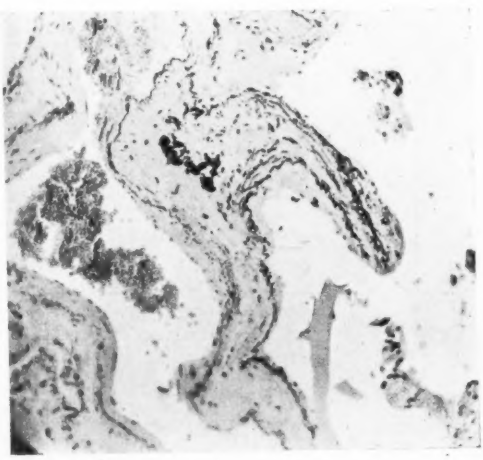
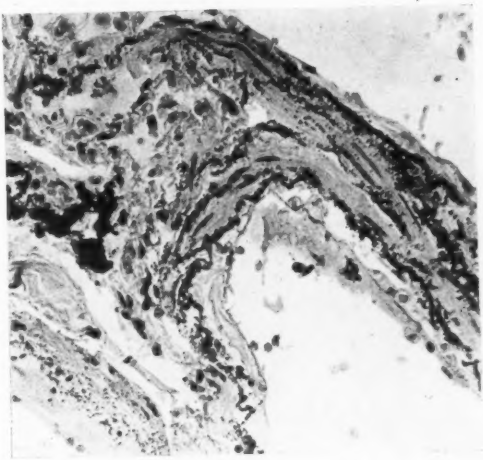


FIG. 3

FIG. 4

FIG. 5





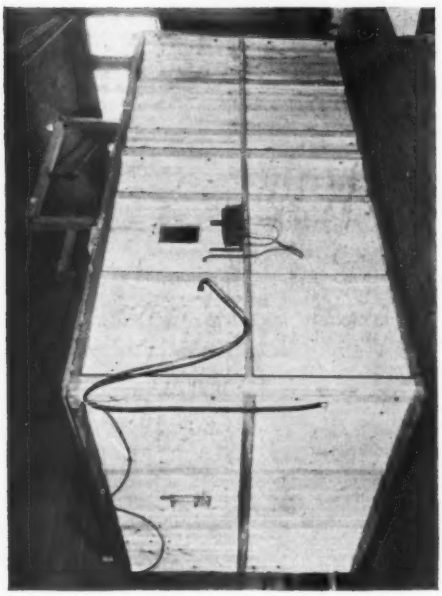


FIG. 1.—Trial room

FIG. 3.—Stowage section

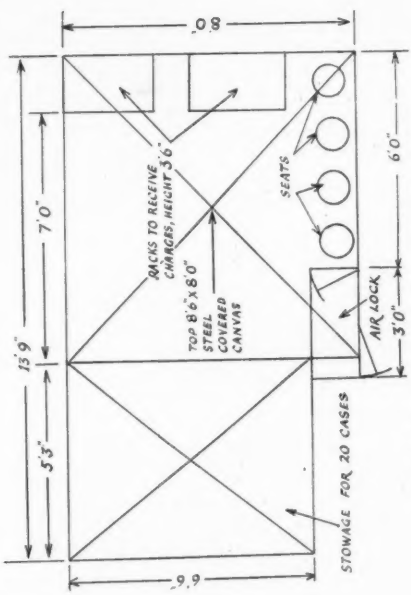


FIG. 2.—Plan of structure. Height 6 ft. 8 in.

FIG. 1. Radiograph of pelvis of furnaceman aged 37. He had worked on open-crucible furnaces for 5½ years.

FIG. 2. Radiograph of lumbar spine of the same man.

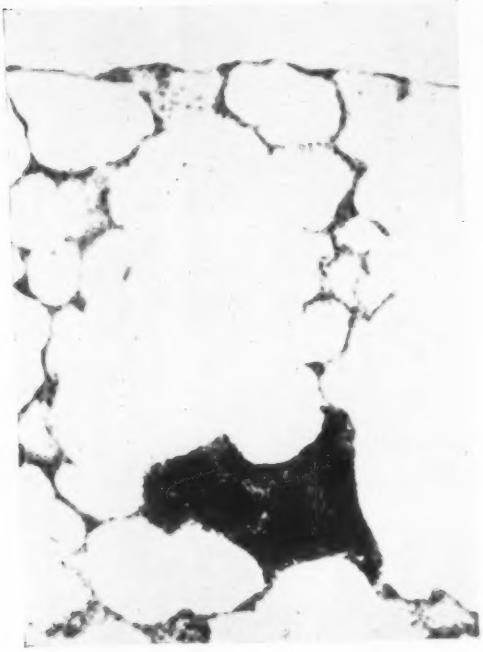


FIG. 1.

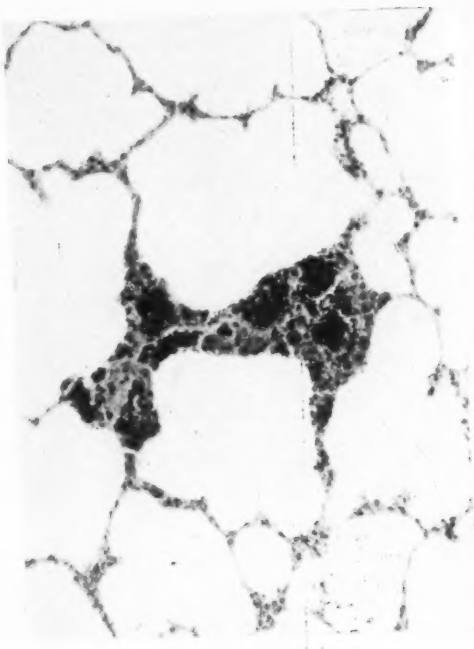


FIG. 2.

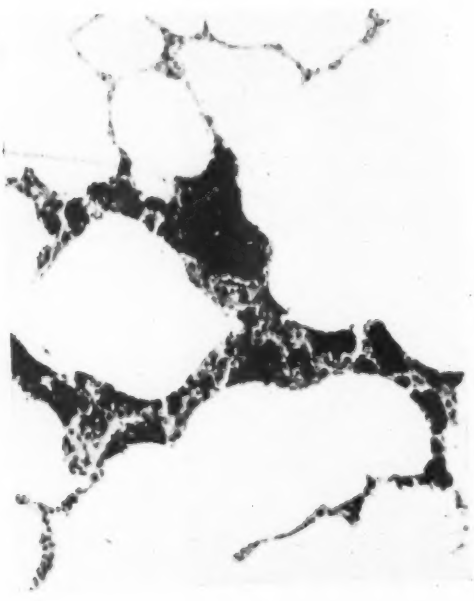


FIG. 3.

FIG. 4.

FIG. 1.—Aggregation of dust-filled phagocytes a short distance beneath the pleura. Hæmatoxylin and eosin × 190.

FIGS. 2 and 3.—Aggregates of dust-filled phagocytes within lung. Hæmatoxylin and eosin × 190.

FIG. 4.—Radiograph of lungs, natural size. The heart was removed before photographing.

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THE SOLUBILITY OR DISTRIBUTION COEFFICIENT OF TRICHLORETHYLENE IN WATER, WHOLE BLOOD, AND PLASMA

BY

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(RECEIVED FOR PUBLICATION, AUGUST 15, 1947)

In a previous paper (Powell, 1945a) the elimination of trichlorethylene and its metabolite, trichloracetic acid, was studied. The present investigation is concerned with the solubility properties of trichlorethylene, upon which its initial absorption and transference in the body depend.

Methods

To determine the solubility of trichlorethylene in water, whole blood, and plasma, similar techniques were used to those employed in determining the solubility of carbon tetrachloride (Powell, 1945b). Trichlorethylene-air mixtures were made up in gas cylinders under pressure. The trichlorethylene concentration varied between 500 and 8,000 vapour parts per million, that is, 0·26 to 3·9 mg. per 100 ml., the maximum pressure allowable being calculated from the saturated vapour concentration at 20° C. To produce higher concentrations, an Oxford vaporizer was used (Epstein and others, 1941).

Water was equilibrated with gas mixture by a simple bubbling technique. For the equilibration of blood and plasma, an apparatus containing a rotating tonometer was used. Blood was obtained from normal donors, unless otherwise stated, oxalate being used as an anticoagulant. Hæmoglobin was measured by Haldane's carboxyhæmoglobin method. Plasma proteins were determined by the micro-Kjeldahl technique, and plasma fats by the method of Bloor (1928).

A slight modification was introduced in the estimation of trichlorethylene by treatment with pyridine and alkali (Habgood and Powell, 1945). It was found that the reaction could be made rather more sensitive by adding 1 ml. of 1 per cent. hydrochloric acid to the final orange-coloured solution. This gave a yellow colour which was measured with a filter transmitting at 430 m.u.

Results

The results obtained by equilibrating water with trichlorethylene-air mixtures at 20° C. and 37° C. are shown in Table 1. The trichlorethylene absorbed is directly proportional to its concentration in the gas, the Ostwald solubility, or distribution coefficient, being $3{\cdot}0 \pm 0{\cdot}1$ at 20° C., and $1{\cdot}55 \pm 0{\cdot}05$ at 37° C. This relationship is maintained from a concentration of 0·26 mg. per 100 ml. to saturation.

As may be seen from Tables 2A and 2B, the distribution coefficient for whole blood varies between 18 and 22 at 20° C., and between 8 and 10 at 37° C. There is no direct relationship between hæmoglobin content of whole blood and its trichlorethylene absorption. Three blood samples of hæmoglobin 64 per cent., 90 per cent., and 110 per cent., were equilibrated at 22° C. with air containing

TABLE 1

SOLUBILITY OF TRICHLORETHYLENE IN WATER AT 20° C. AND 37° C.

Trichlorethylene (mg./100 ml.)			$\frac{C_{Water}}{C_{Air}}$	
Air	Water			
	20° C.	37° C.	20° C.	37° C.
0·26	0·77	0·42	3·0	1·6
0·55	1·70	0·90	3·1	1·6
1·08	3·12	—	2·9	—
1·23	3·75	1·78	3·1	1·5
2·10	6·38	—	3·0	—
2·22	6·88	3·72	3·1	1·6
11·0	34·5	—	3·1	—
15·8	49·5	—	3·1	—
†43·0	127	†122	3·0	1·5

† Concentration of trichlorethylene in saturated vapour at 37° C. is 84 mg. per 100 ml.

* With a grant from the Medical Research Council.

0·55 mg. per 100 ml. The distribution coefficients were 19, 21, and 19 respectively. Further examples of this kind can be seen in Tables 2A and 2B.

It was found, however, that solutions of hæmoglobin in water absorbed trichlorethylene in proportion to their hæmoglobin content. To prepare these hæmoglobin solutions, washed erythrocytes were lysed with water and centrifuged at high speed to bring down cell envelopes and stroma. The results obtained with these solutions are illustrated in the figure. It was also found that red cell constituents other than hæmoglobin do not influence the uptake of trichlorethylene. Thus, a washed cell suspension of hæmoglobin 86 per cent. absorbed 3·12 mg. per 100 ml. in contact with air containing 0·28 mg. of trichlorethylene per 100 ml. An equivalent hæmoglobin solution prepared in the above manner absorbed 3·33 mg. of trichlorethylene per 100 ml. from the same gas mixture.

In Table 3 the solubility of trichlorethylene in whole blood is compared with that in plasma. It is seen that red cells absorb relatively more trichlorethylene than does plasma, the distribution coefficient in the latter case ranging from 15 to 20.

TABLE 2A

SOLUBILITY OF TRICHLORETHYLENE IN BLOOD AT 20° C. AND 37° C.

Donor	Trichlorethylene (mg./100 ml.) In Air	In Blood		$\frac{C_{Blood}}{C_{Air}}$	
	0·55mg./100ml.) 1,000 v.p.p.m.	20° C.	37° C.	20° C.	37° C.
(1.) Hæmoglobin 122%	0·28	5·30	2·55	19	9·1
	0·56	11·3	—	20	—
	1·10	20·7	11·3	19	10
	2·06	41·0	20·8	20	10
(2.) Hæmoglobin 98%	0·28	5·85	2·41	21	8·7
	0·56	12·0	5·75	21	10
	1·10	23·0	9·94	21	9·0
	2·06	41·2	18·5	21	9·0
(3.) Hæmoglobin 88%	0·26	4·65	—	18	—
	0·55	10·6	4·30	19	7·8
	1·08	21·0	9·70	20	8·8
	2·10	39·0	—	19	—

TABLE 2B

SOLUBILITY OF TRICHLORETHYLENE IN BLOOD AT 20° C.

Donor	Trichlorethylene (mg./100 ml.) Air	Blood	$\frac{C_{Blood}}{C_{Air}}$
4. Hæmoglobin 118%	0·29	6·10	21
	0·60	13·3	22
	1·10	22·8	21
	17·1	36·0	21
5. Hæmoglobin 110%	1·23	23·1	19
	2·22	43·3	19
	5·28	95·6	18
	10·7	194	18

TABLE 3

SOLUBILITY OF TRICHLORETHYLENE IN BLOOD AND PLASMA AT 20° C.

% Hb (Haldane)	Trichlorethylene (mg./100 ml.) Air	Blood	Plasma	$\frac{C_{Blood}}{C_{Air}}$	$\frac{C_{Plasma}}{C_{Air}}$
64	0·55	10·2	9·40	19	17
118	0·30	6·10	5·95	20	20
122	0·28	5·30	4·65	19	17
	2·06	41·0	39·0	20	19
88	1·08	21·0	15·9	20	15
	2·10	39·0	34·3	19	16
112	5·28	95·6	85·3	18	16
	10·7	194	180	18	17
98	0·56	12·0	10·3	21	18
	1·10	23·0	19·0	21	17
	2·06	41·2	37·6	20	18

TABLE 4

SOLUBILITY OF TRICHLORETHYLENE IN PLASMA AT 20° C.

Donor	Plasma fat (mg./100 ml.)	protein (g./100 ml.)	Trichlorethylene (mg./100 ml.) Air	Plasma	$\frac{C_{Plasma}}{C_{Air}}$
Normal ..	570	7·2	2·13	42·4	20
Normal ..	594	7·0		42·6	20
Anæmia ..	480	7·9		46·0	22
Leukæmia ..	510	6·0		30·5	14
Anæmia ..	386	5·5		30·0	14
Normal ..	720	8·1	0·55	12·0	22
Normal ..	580	7·0		10·3	21
Sprue ..	590	6·0		9·36	17
Ileitis ..	360	7·3		9·01	16
Normal ..	770	7·4	0·26	5·40	21
Neoplasm ..	710	7·2		5·36	21
Diabetes / Nephritis	1,020	5·2		4·51	18
Leukæmia ..	786	4·9		4·35	17
Liver damage	504	5·6		3·70	14
Nephrosis ..	620	5·1		3·15	12

TABLE 5

SOLUBILITY OF TRICHLORETHYLENE AND CARBON TETRACHLORIDE IN FAT EMULSIONS AT 20° C.

	Oleyl alcohol (450 mg./100 ml.)		Oleic acid (1,000 mg./100 ml.)		Arachis oil (900 mg./100 ml.)	
	Chlorhydrocarbon absorbed (mg./100 ml.)	Distribution coefficient	Chlorhydrocarbon absorbed (mg./100 ml.)	Distribution coefficient	Chlorhydrocarbon absorbed (mg./100 ml.)	Distribution coefficient
Carbon tetrachloride-air mixture (0·72 mg./100 ml.)	8·58	12	18·2	25	14·0	20
Trichlorethylene-air mixture (0·55 mg./100 ml.)	3·33	6·0	6·50	12	8·10	15

An attempt was made to determine whether the trichlorethylene distribution coefficient of plasma was related to its protein and fat content. The data given in Table 4 were used in the calculation, giving a value for b_1, the partial regression coefficient for trichlorethylene solubility on protein of $2{\cdot}750 \pm 0{\cdot}369$. The corresponding figure b_2 for fat was $0{\cdot}00963 \pm 0{\cdot}00233$. The value for b_1 would occur by chance in less than 1 in 1,000 times, and that for b_2 in less than 1 in 100 times. The trichlorethylene absorption of plasma is almost certainly dependent, therefore, on its protein content and probably on its fat content.

This conclusion is supported by the results obtained on equilibrating fat emulsions in water at 20° C. with a gas mixture containing 0·55 mg. of trichlorethylene per 100 ml. Emulsions of oleyl alcohol, oleic acid, and arachis oil were used—these substances being considered as models for plasma cholesterol, fatty acid, and neutral fat respectively. No attempt could be made to assess the absorption of phospholipids, owing to the chemical instability of these compounds. The results are summarized in Table 5, together with a set of corresponding values for a carbon tetrachloride-air mixture containing 0·72 mg. per 100 ml. The distribution coefficients found for trichlorethylene were low compared with those given by plasma of equivalent fat content. On the other hand carbon tetrachloride-air mixture equilibrated with oleyl alcohol gave a distribution coefficient comparable with that of normal plasma. The more concentrated oleic acid and arachis oil emulsions gave very high distribution coefficients with carbon tetrachloride. Unfortunately no figures are available for comparison of these latter figures with those given by plasma containing an equivalent amount of fat. Assuming that carbon tetrachloride absorption is proportional to the amount of fat present, oleic acid and arachis oil at lower concentrations will give roughly the same distribution coefficients as the oleyl alcohol emulsion.

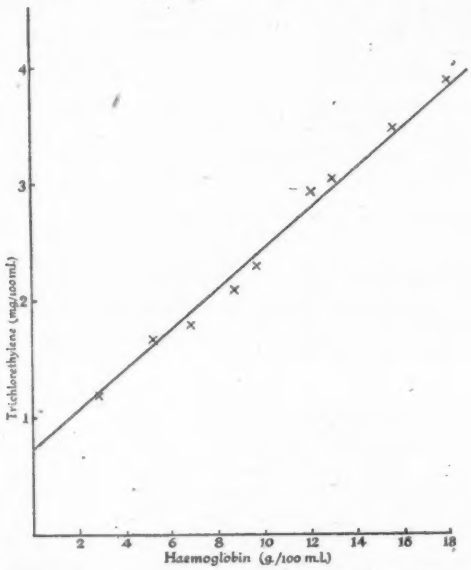
Discussion

Trichlorethylene is more soluble in water than carbon tetrachloride, but less soluble than chloroform. In the case of whole blood, the distribution coefficient of both trichlorethylene and chloroform (Nicloux, 1929) is in the region of 20 at 20° C., and 9 at 37° C.—values markedly higher than those obtained for carbon tetrachloride (Powell, 1945b). This may be accounted for partly by the affinity of trichlorethylene and chloroform (Scotti-Foglieni, 1930) and the non-affinity of carbon tetrachloride, for hæmoglobin. Both chloroform and trichlorethylene are rather less soluble in plasma than in whole blood, this being in contrast with carbon tetrachloride, where the distribution coefficient at 20° C. is in the region of 4·5 for whole blood and 9 for plasma.

The results obtained with fat emulsions indicate that the plasma fat concentration cannot wholly account for the amount of trichlorethylene absorbed. Taking into consideration the affinity of trichlorethylene for hæmoglobin, it is reasonable to assume that the plasma proteins are also concerned with its uptake. The correlation between the distribution coefficient of plasma and its fat and protein content tends to confirm this.

In the case of carbon tetrachloride, fat emulsions roughly equivalent in fat content to normal plasma give distribution coefficients of 12. It seems, therefore, that plasma fat can wholly account for the carbon tetrachloride taken up, and that plasma

proteins and hæmoglobin do not influence the absorption. It seems possible that the affinity of all three chlorhydrocarbons for plasma fat involves a simple solution process, while the attachment of trichlorethylene and chloroform to protein may depend on an absorption process for which the non-polar carbon tetrachloride molecule is particularly unsuited.



Solubility of trichlorethylene in hæmoglobin solutions at 20° C.

Summary

The distribution coefficient of trichlorethylene between water and air is 3·0 at 20° C. and 1·6 at 37° C. The air concentration used varied between 0·26 and 15·8 mg. per 100 ml.

The distribution coefficient between blood and air is constant in a given specimen of blood throughout the above concentration range. The coefficient in a series of specimens ranged between 18 and 22 at 20° C., and between 7·8 and 10 at 37° C.

Hæmoglobin solutions in water absorb trichlorethylene in proportion to their hæmoglobin content.

The distribution coefficient of trichlorethylene between plasma and air varies between 16 and 20 at 20° C.

Evidence has been obtained that the absorption of trichlorethylene by plasma is correlated with its fat and protein content.

The absorption of carbon tetrachloride by the blood probably depends on the concentration of plasma fat alone, and is uninfluenced by plasma proteins and hæmoglobin.

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References

Bloor, W. R. (1928). *J. Biol. Chem.*, **77**, 53.
Epstein, H. G., Macintosh, R. R., and Mendelssohn, K. (1941). *Lancet*, **2**, 62.
Habgood, S., and Powell, J. F. (1945). *Brit. J. industr. Med.*, **2**, 39.
Nicloux, M., and Scotti-Foglieni, L. (1929). *Ann. de Physiol.*, **5**, 434.
Powell, J. F. (1945). *Brit. J. industr. Med.*, **2**, 142.
—— (1945). *Ibid*, **2**, 212.
Scotti-Foglieni, L. (1930). *C. R. Soc. Biol. Paris*, **105**, 959.

Brit. J. industr. Med., 1947, **4**, 237.

BARRIER CREAMS AND THEIR EVALUATION

BY

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In view of the recent publication by Sadler and Marriott (1946) on the evaluation of barrier creams, it may be of interest to describe some work carried out during the 1939–45 war. Whilst the work herein described was mainly concerned with explosives, it illustrates the line of attack which might be adopted in dealing with one aspect of the prevention of industrial dermatitis. In the earlier years of the war some anxiety was expressed with regard to the extent of dermatitis in certain factories dealing with explosives. Whilst the highest incidence of dermatitis occurs with tetryl, other nitrobodies, such as T.N.T., are not above suspicion. Tetryl dermatitis has been ascribed by some authorities to a mechanical effect produced by the acicular crystalline form of this product in similarity to the dermatitis produced in many industrial operations. Other authorities claim that the effect is due to absorption of the tetryl by the epidermis of the skin. Experience indicates that the latter is the more likely cause, for dermatitis is often frequent where the material is not in the crystalline form. Tetryl differs from T.N.T. in that the former was prepared finally in dry form whereas T.N.T. was handled mostly molten before being mixed with other substances. Tetryl dermatitis was much more frequent, therefore, in filling than in explosives factories, where in the great majority of operations it was handled wet.

A paper by Witkowski and others (1942) quotes results of an enquiry made at an American factory where 1,258 operatives were employed on tetryl: 944 suffered from dermatitis, though only three of the cases required hospital treatment. In order to control the amount of dust they recommended that the crystalline tetryl should be blended with stearic acid and that granular tetryl (still crystalline but of different form) should be blended with graphite. The authors of that paper developed a barrier, full details of which are not given, and claim that its use cut down the weekly incidence of dermatitis by 50 per cent.

Attention may also be drawn to a paper by Swanston (1943), in which statistics are given of dermatitis in this country. In June, 1942, 3·3 per cent. of the total contact workers were reported as cases of dermatitis, of which one quarter were off work and receiving compensation. In May, 1943, the figure was reduced to 1·5 per cent. Of the total incidence, tetryl dermatitis represents more than one half, mercury fulminate approximately one-third, the remainder being caused by T.N.T., ammonium nitrate, shellac, picric shellite, lead azide, etc. For all practical purposes, the irritant explosives are mercury fulminate and tetryl. T.N.T. may be added not so much because of its high incidence, but rather on account of the severity of the dermatitis produced by it. Dust seems to be the cause of irritation in the majority of cases.

The Present Inquiry

Early in the war a questionnaire was sent to all medical officers at factories in the hope of gaining some precise knowledge about the incidence of dermatitis and experience of the use of barriers. A statistical study was made difficult by the fact that the quantity of explosives manufactured and handled varied in different factories. Furthermore, at one stage in the war there was a large influx of female labour. In some factories barriers were already in use, while in others the most important factor controlling dermatitis was claimed to be personal hygiene.

From the results of the questionnaire it was possible to make the following deductions: (1) women are more disposed to use barriers than men, and the percentage of dermatitis amongst the latter is higher; (2) face and hands are involved, and contact with air-borne and dry solid explosives seems to be the main cause; (3) barriers are used at least twice per eight-hour shift.

It would appear therefore, that the case for the use of barriers is justified. In connexion with tetryl dermatitis it is worth noting that the mechanism of

the interaction is believed to be due to the effect of light on the tetryl once it has gained attachment to the skin surface. Brownlie and Cumming (1946) have shown that tetryl is quantitatively converted into a well-known strongly dermatitic agent, methyl picramide, on exposure for a few hours to ultra-violet irradiation, and that the product is ultimately decomposed by ultra-violet light. Support is given to this work by the fact that tetryl dermatitis reaches its highest incidence in about sixteen days and is claimed to be more prevalent in hot sunny weather. Moreover, it suggests that ultra-violet irradiation of the skin when œdema has become apparent would be beneficial.

In order to assess the amount of nitrobody dust and vapour in the atmosphere of explosives factories, methods have been worked out (Cumming and Wright, 1945). As a result of frequent surveys it was possible to improve considerably the indoor atmospheric conditions by modifications in ventilation etc., thereby reducing the risk of contamination of the skin during normal working operations.

Barriers in Use at the Time of the Survey

At this stage it was decided to test the barriers which were already in use at certain factories. To do this it was necessary to devise a simple test. This consisted in applying the barrier to the skin of the forearm after thorough cleansing of the surface with soap and warm water, and drying. The barrier was rubbed in to the skin so as to give a uniform film. The same operatives conducted all tests in the first instance so as to eliminate, as far as possible, the personal element. Preliminary tests on a large number of persons proved that the thickness of film normally applied to the skin is remarkably constant and varied slightly from 20 μ. When the film had been thus produced, powdered T.N.T. or tetryl was dusted on to the surface, the surplus shaken or blown off, and the arm exposed to the atmosphere for one to three hours under normal working conditions. After this exposure, the coating on the skin was removed by thorough washing in warm water and soap. The surface of the skin was not dried, nor touched in any other way. A swab of cotton wool moistened with sodium sulphite solution was then rubbed over the surface. If the cotton wool took on a pink colour, the test was taken to be positive, namely, that the

TABLE 1

RESULTS OF TESTS ON BARRIER CREAMS IN USE AT CERTAIN FACTORIES

Sample	Reaction	Characteristics	Positive penetration hours	
			T.N.T.	Tetryl
A	Alkaline	Slightly greasy	1	1
B	,,	Greasy	1	1
C	,,	Slightly greasy	1	1
D	Neutral	Greasy	1	1
E	Alkaline	,,	1	1
F	,,	,,	1	1
G	,,	Slightly greasy	1	1
H	,,	Greasy	1	1
J	,,	,,	1	1
K	,,	Greasy and gritty	3	3
L (i)	,,	Greasy	1	1
(ii) powder	,,	—		
M (i)	Neutral	Greasy	1	1
(ii) powder	,,	—		
N varnish	Acid	Non-greasy	1	1
O varnish	,,	Slightly greasy and gritty	1	1
P varnish	,,	Greasy	1	1
Q	Neutral	Slightly greasy	1	1
R (i)	Acid	Greasy	1	1
(ii) powder	Alkaline	—		
S	,,	Greasy	1	1
T	Neutral	Slightly greasy	1	1
U varnish	,,	Non-greasy	1	1
V	Alkaline	Slightly greasy	1	1
W	,,	Greasy	3	3
X	,,	,,	1	1
Y	,,	Slightly greasy	1	1
Z	Acid	Greasy	1	1

TABLE 2

SOLUBILITIES G/100 G. SOLVENT

Substance	T.N.T. at 35° C. or temp. stated	Tetryl at 35° C. or temp. stated	Picric Acid at 35° C. or temp. stated	Hexamine at 35° C. or temp. stated
Liquid Paraffin	0·01	0·01	Negligible	0·08
Glycerine	0·10	0·2	0·40	4·10
Castor oil	0·50	3·4	0·10	1·9
Paraffin wax	0·4 (58°)	0·04 (65°)	0·04 (60°)	0·16 (65°)
Cocoa butter	0·6	0·3	0·1	0·48
Lanoline	2·7 (80°)	0·4 (65°)	2·6 (80°)	23·2 (65°)
Lanette wax	0·9 (80°)	0·4 (75°)	0·4 (80°)	2·3 (75°)
Palmitic acid	1·0 (64°)	0·3 (65°)	Negligible (65°)	13·7 (75°)
Stearic acid	1·0 (70°)	0·3 (70°)	Negligible (20°)	11·3 (70°)

nitro-compound had penetrated through the barrier to the skin surface. If any doubt arose, the test was again repeated in duplicate. When a barrier incorporated the use of a face powder on a vanishing cream ground, both were applied in the customary manner before the nitrobody was dusted on to the surface.

In addition, the barriers were tested for reaction to litmus and the characteristics (greasiness etc.) were agreed upon by four independent observers after application to the skin and rubbing on the surface.

The results of these tests are given in Table 1.

The following conclusions may be drawn.

(*a*) All barrier creams examined were greasy or slightly greasy in character. The only non-greasy types occurred in the skin varnish group.

(*b*) Sixty-five per cent. were alkaline in reaction and, of the remaining 35 per cent., less than half were acid in reaction, these almost invariably occurring in the skin varnishes.

(*c*) With only two exceptions, the barriers were penetrated by both T.N.T. and tetryl in one hour. These two barriers were, however, penetrated in three hours by both nitrobodies.

It is clear from the results quoted that none of the barriers in use at the time was efficient. Protection must be given for some hours before their efficiency can be judged to be satisfactory from the practical point of view.

The Ideal Barrier

The ideal barrier would be a material of the appropriate physical form and composition in which the nitrobodies encountered would be insoluble, and the degree of absorption of the barrier by the skin would be at a minimum. As stated above, the thickness of film produced was found to be about 20 μ, which is in accord with the figure mentioned by Sadler and Marriott. The particle size of tetryl, as of many other dusts, which is most physiologically active is 5 μ and under (Bedford and Warner, 1942). No material of this size could, therefore, penetrate the film to the skin surface. For safety the consistency of the barrier should be such as to allow of a film of this order of thickness being obtained in the normal method of application.

It was impossible to determine the solubility of the nitrobodies directly in the barrier cream, but determinations were made in the components which are known to be the main basis of creams and vanishing creams. The results are given in Table 2. In addition to T.N.T. and tetryl this includes picric acid and hexamine, since in the questionnaire referred to above it was suspected in one or two cases that dermatitis had been caused by these compounds. Mercury fulminate is not included in the table, as only in lanoline did it show any detectable solubility.

The components were treated with excess of the dermatitic agent and maintained at a constant temperature for one hour. After separation of the excess, acetone (for tetryl) or alcohol (for T.N.T.) was added to dissolve, and the coloration produced with alkali, compared against a standard, gave the required result. In the case of picric acid, potassium cyanide was used as the colour reagent. Hexamine was determined quantitively by hydrolysing with standard acid and back titrating with standard alkali.

The results prove conclusively that the products examined possess high degrees of solubility in components of vegetable or animal origin. Noteworthy is the high solubility in lanoline of T.N.T., picric acid, and hexamine. Only in the case of liquid paraffin was a very low solubility in general recorded. For all the bodies examined paraffin wax was also satisfactory, except perhaps in the case of T.N.T. Even in this case the solubility is higher than would be encountered at skin temperature. It follows, therefore, that lanoline might be used in a tetryl barrier but it would be inadmissible in a barrier designed to protect against the other products. Likewise, castor oil would be satisfactory for T.N.T. and picric acid but not for tetryl.

Vegetable Oils, which are the main constituents

of vanishing creams, are to be avoided in barriers for nitrobodies. In view of their marked contrast in constitution from the fats present in the skin, paraffinoids will not be readily absorbed by the surface. In view of the action of alkali on the skin and on its interaction with nitrobodies to form highly coloured soluble salts which may be toxic, it is considered that the best barrier will be neutral in reaction, at any rate not alkaline.

Attention has already been drawn to the importance of maintaining a film on the skin of about 20 μ thickness, so as to avoid airborne crystalline tetryl of size 5 μ from making contact with skin by piercing the film. Obviously the thickness of film produced must be determined by the viscosity of the cream at the skin temperature. It is, therefore, suggested that the softening point of the cream should be about 22° C. This latter fact has also some psychological importance. It is held that if protection is to be guaranteed for some hours, then the worker should appreciate that it takes a few minutes to ensure that an even film has been applied to the whole of the exposed skin surface. The film must not be patchy, and a fairly high softening point ensures that the worker will take time to protect himself, particularly if he has confidence in its protective powers.

It is important that a barrier should be capable of removal by the ordinary processes of soap and water. It is suggested, however, that it should not be too easily removed. It should require considerable amounts of hot water and soap. This helps to prevent the transfer of dangerous dust outside the factory into homes where contact may be made with other persons and with food. An emulsifying agent of the water-soluble type must, therefore, be present.

It will be noted that a barrier cream based on paraffinoid compounds will give protection from all the explosive substances mentioned.

The Camraild Barrier

Based on the above conditions a new barrier was developed and known during the war as " Camraild barrier." It consisted of a mixture of paraffin wax m.p. 135° F. (20 per cent.), ordinary vaseline (40 per cent.), lanette wax, as emulsifying agent (10 per cent.), and water (30 per cent.). The mixture is made up as follows.

The paraffin wax, lanette wax, and vaseline are melted together, keeping the temperature as low as possible. The mixture is cooled to 50° C. and the water, after heating to the same temperature, added with vigorous stirring until the cream is of uniform consistency. Perfume may be added if desired.

The barrier so prepared tends to have a greasy feel, but if applied to the skin in proper amount and well rubbed in it does not prevent use of the hands for normal manual work. In any case over 60 per cent. of the barriers in use during the war were described as greasy (Table 1). But even then, slight greasiness must not be held against a barrier if it can be proved experimentally that it is efficient for the purpose intended.

It will be recalled (Table 1), that only two of the barriers tested gave protection from tetryl or T.N.T. for three hours, the others all failing in one hour. Accordingly, a test was made with Camraild barrier on ten different observers using the simple technique described on p. 238. Having shown that a negative test was obtained in three hours, the test was extended to six hours and in every case a negative result was recorded for both tetryl and T.N.T. When solid material such as zinc oxide, boric acid were incorporated the results were variable and penetration was found in about 50 per cent. of the tests over a three-hour period. Furthermore, these solids tended to give a gritty character to the cream.

Evaluation

The test already described is not free from criticism. The personal factor cannot be neglected. For this reason the tests were applied to as large a number of observers as possible. Nevertheless, it was considered desirable to place the test on a more scientific basis, in which the same thickness of film would be tested with the same amount of nitrobody under conditions of controlled humidity.

It is obvious that to prepare a film of 20 μ thickness for test is impracticable. It was agreed after experiment that 1 mm. was easily prepared, and although the tests would not bear the same relationship to those obtained from the previous practical test they would at least be comparable. It was also considered desirable to use the same amount of nitrobody and to expose this over the same area. This was done by using a known and constant volume of a solution of the nitrobody in acetone and allowing the solvent to evaporate at atmospheric temperature over a known area. The solution deposited a film of minute crystals so that unit weight per unit area was always constant.

The test was first of all carried out at 30° C. However, as several of the creams lost their original texture and became quite hard on exposure at this temperature for more than an hour or two, a lower temperature was adopted. To prevent loss of moisture during the test, the creams were kept exposed to an atmosphere of between 90 and 95 per cent. humidity. The reagent used was strong aqueous KOH and, in order to reduce the possibility of penetration of this into or through the barrier, the time of test was limited to a maximum of one minute. In this new test, the nitrobody penetrates upwards through the barrier.

The procedure adopted is described below, and in Table 3 are given the results applied to some barriers already referred to, as well as to some later products. It will be seen that the results are in general agreement with those reported, and that Camraild barrier is the most efficient protective agent against both T.N.T. and tetryl.

Penetration Test for Barrier Creams.—A brass ring about 1 in. diameter, 1·5 in. external diameter, and 1 mm. thick is placed on a carefully cleaned ground-glass surface. The inner circumference of the ring is traced with pencil on the glass. The ring is removed and the circular area on plate is covered with 0·1 c.cm. of nitrobody solution. The concentrations used in these tests were 5 g. nitrobody to 50 c.cm. acetone. The solvent is allowed to evaporate in air. The ring is replaced as before and the mould so formed filled with the cream under test, the surface of the cream being smoothed and levelled with the sides of the ring by means of a straight-edge.

The test equipment is then placed in a chamber kept at between 20° and 22° C., the atmosphere in the chamber being maintained at 90 to 95 per cent. humidity by passage of air previously bubbled through wash bottles of warm water, and using a wet-bulb thermometer inside the chamber. The creams are removed periodically and the surface touched with a small drop of 40 per cent. aqueous KOH. The appearance of a pink colour *within* one minute of applying alkali is taken to indicate penetration of layer by nitrobody. One specimen allows of three to four tests with alkali being carried out. When a positive is obtained, it should be checked on a fresh specimen. That duplication of time of penetration is attainable without difficulty has been established after a large number of observations.

When an emulsified non-absorbable cream is applied to the skin, it may be assumed that (*a*) water is absorbed by the skin, leaving a film of the actual barrier substance, or (*b*) water is lost by evaporation from the cream film. In the case of (*b*), it may well be that the rate of evaporation of water from the film of cream may be compensated by removal of water from the skin to the film, an equilibrium being thus established.

TABLE 3

PENETRATION (IN HOURS) OF T.N.T. AND TETRYL THROUGH BARRIERS 1 MM. IN THICKNESS AT 20 TO 22°C. AT A HUMIDITY OF 90 TO 95 PER CENT.

Sample	T.N.T.	Tetryl
A_2	pos. 2	pos. 2·5
B_2	pos. 24	pos. 20
C_2	pos. 1	pos. 2
H	pos. 2	pos. 2·5
Q	pos. 6	pos. 16
T	pos. 2	pos. 1
W	pos. 3	pos. 3
Camraild	neg. 24	neg. 20

Note on Lanoline.—Penetration tests were also carried out on lanoline. To obtain a film of this substance, it was necessary to raise its temperature to 30° C. and to maintain this temperature during test. No control was kept on the humidity of atmosphere as there should be little loss of moisture from lanoline. The result was: T.N.T., pos. 16; tetryl, neg. 16. This result is in accord with the solubilities of these nitrobodies in lanoline already reported.

The authors desire to thank the Explosives Directorate of the Ministry of Supply for permission to publish this paper.

REFERENCES

Bedford, T., and Warner, C. G. (1942). *J. industr. Hyg.*, **24**, 315.
Brownlie, I. A., and Cumming, W. M. (1946). *Biochem. J.*, **40**, 20.
Cumming, W. M., and Wright, W. G. D. (1945). *Brit. J. industr. Med.*, **2**, 83.
Sadler, C. G. A., and Marriott, R. H. (1946). *Brit. med. J.*, **2**, 769.
Swanston, C. (1943). *Proc. roy. Soc. Med.*, **36**, 633.
Witkowski, L. J., Fischer, C. N., and Murdock, H. D. (1942). *J. Amer. med. Ass.*, **119**, 1406.

BOOK REVIEWS

THE SCIENTIFIC AND SOCIAL WORK OF THE NATIONAL INSTITUTE OF INDUSTRIAL MEDICINE AND HYGIENE OF SPAIN

In this booklet there is a preliminary review of the hygiene and health measures which were introduced in Spain in early times. In 1342 Pedro II took steps to deal with malaria. James I laid down the guiding principles for the construction of drains and the provision of a pure water supply, and in 1751 Ferdinand VI made the notification of tuberculosis obligatory. Since those times the number of regulations has increased, and in the last century the government's interest in industrial health was manifested on many occasions.

In 1932 a revised code covering accidents was drawn up. More attention to industrial conditions was apparent in the medical schools, and in July, 1944, a decree brought about the foundation of a National Institute of Industrial Medicine, Hygiene, and Safety. In this institute courses of instruction in industrial diseases are regularly given and ideas are spread by continuous propaganda. The institute works in collaboration with the medical profession, insurance societies, the ministry of social welfare, and the courts dealing with compensation cases.

In the book under review it is suggested that the Spanish artisan and craftsman was unequalled in Europe at the time of the expulsion of the Moors and the freeing of the slaves. At a later date, when wealth from overseas flowed into Spain, there was less progress in manufacture and invention. The landowners were too rich to feel any material need to foster progress in these directions, and also too proud to soil their hands with trade. During the nineteenth and early twentieth centuries Spain lost ground that is only now being slowly recovered.

This institute has begun investigations into the hygienic conditions of forest workers and workers in lead, in the printing trade, and in other employment. It is also recommending that the list of trades in which women and children should not work might be extended.

The official interpretation of an industrial accident is in Spain so wide as to cover all forms of illness and all injuries attributable to employment. As industry has not been very extensive, the number of recognized industrial illnesses has not been as great as elsewhere. Nevertheless, in 1936, the Cortes drew up a list of industrial disorders which has formed the basis of more recent legislation. Particular attention was given to pneumoconiosis. Orthopædic surgery and the surgical treatment of accidents has not received as much attention in Spain as seems necessary. The institute is taking active steps to remedy this defect and to arrange for the necessary experimental work, the manufacture of artificial limbs, means of assistance and retraining for the disabled, and a close link with the lawyers handling compensation cases.

An industrial museum is being formed in which special displays will draw attention to the methods of preventing accidents and industrial diseases. In connexion with this a laboratory and research service is organized and a statistical office has also been set up.

[This pamphlet of 56 pages is necessarily only a brief outline. Since the measures described are of recent origin it appears unlikely that the institute can produce much original work in the near future.] *G. C. P.*

DYNAMIC MOTION AND TIME STUDY

By James J. Gillespie
(Paul Elek (Publishers) Ltd., London. 1947. Pp. 95. Eight illustrations. Price, 7s. 6d.)

The presentation of Gillespie's monograph loses nothing by being unorthodox. There can be no mistake about his opinions; such forthright language is a pleasure after the usual mealy-mouthed agreements.

The author is unquestionably right to quarrel with the attitude of mind which causes some advocates of time and motion study to measure operation time cycles to the fifth decimal place of a minute. This he dubs "micromotionism." Equally right is the author's stress that the operator and the operator's response and prejudice are the most important element in any factory operation. At least here is a glimmering that the functions of a human being are not so simple as job sheets. Rearrangement of jobs to avoid unnecessary effort does good to all but, as is hinted, the wisdom of attempts to dictate the manner and time of an operation movement is open to doubt. Apart from a few references to Plato, the author takes little account of physiological or psychological knowledge. Recent work (for example by Bartlett) on the psychological aspects of fatigue and skill is entirely ignored. The medical reader will be left with the feeling, in spite of Gillespie's valiant efforts to enlighten soul-searing orthodoxy, that time and motion study is neither art nor science but a dogma.

T. A. L. D.

BRITISH AND FOREIGN OFFICIAL PUBLICATIONS

PSYCHOLOGICAL DISORDERS IN FLYING PERSONNEL OF THE ROYAL AIR FORCE INVESTIGATED DURING THE WAR 1939–45.

(H. M. Stationery Office, London. 1947. Pp. 344. Price 7s. 6d.)

This publication consists of twenty-three reports completed during the 1939-45 war of the psychological symptoms met with in pilots and air-crew. Over 3,000 cases were met with annually, and the papers here collected indicate some of the predominantly psychiatric problems raised. Yet these papers are of far more importance than the term "psychological disorders" might seem to suggest. They raise problems which can be applied directly to industrial medicine.

The personnel involved were officers and men living under discipline, subjected to the gravest of dangers, and yet carrying upon their shoulders the heaviest of responsibilities and sometimes harassed by home worries. It may seem that the conditions of the individual within the factory are very different; yet discipline, danger, and responsibility are there, though they may seem small as compared with Royal Air Force conditions. We would wish for one loyalty in the factory: we know that there often are two at least, one to the self, one to the workman's union, and little enough to spare for what we will call social responsibilities. In the assessment of psychiatric casualties, absence of this latter loyalty would count as a predisposing factor towards psychological illness. Such illness may be frank states of loss of confidence, anxiety neuroses, or hysteria such as occurred in the Royal Air Force. Such conditions arise in the factory, to be added to by those illnesses of a worker's temperament which cause him to become accident-prone, or to be a focus of discontent or misunderstanding, leavening in a malign fashion an otherwise alterable and co-operative loaf.

For these reasons those symptoms which non-medical officers noted as suggestive of too great a strain have value for industrial medicine—the man's facial expression and bearing, his reaction to stresses such as aerobatics, tremors, fidgets, and so on. A good deal of information was conveyed by how the trainee handled his controls, more still by the onset of erratic reactions (not implying grossly erratic behaviour) in his flying. Requests to cease training or frequent attendance in the sick bay suggested emotional tension and uncertainty. Similar reactions occur amongst apprentices in any large organization, and the problems which faced the Royal Air Force in absorbing or rejecting such trainees are to be expected in civilian life.

It was found that 16 per cent. of the psychological casualties might have been detected upon enlistment (the United States figures are higher); but, paradoxically, amongst this number were persons who actually gained distinctions. A policy of total exclusion of the psychologically predisposed would have reduced the fighting strength too far, and the problem of retaining the somewhat predisposed trainee, of treating his fear (or the symptom which resulted from this), and of increasing his morale have obvious pointers for industrial medicine.

The factors determining the individual's capacity to carry out the arduous duties of air-crew work were resolved into (*a*) his own intrinsic character and (*b*) those external conditions associated firstly with flying conditions, secondly conditions on the station, and thirdly personal and domestic considerations. The reports indicate the search that had to be made for optimum length of tours in bomber, fighter, and coastal commands, and the ways in which different machines carried different risks. The maintenance of morale, the dependence of this upon success, reaction to adversity, the character of leaders, the imagination of seniors, are all discussed. Living conditions, physical training, leisure, spacing of effort, all played their part.

Chapters 4 and 16 may be specially recommended. The latter surveys the methods of assessment of psychiatric traits which may indicate unsuitability for flying, and, we may add, "poor bets" for industrial stability. Lack of aggression and persistence, emotional changeability, and immaturity were regarded as more serious pointers to instability than were anxiety, morbid fears, or obsessional tendencies. Yet here a direct carrying over of Royal Air Force problems to those affecting factories should not be made. The apprentice is under 21, the average age of air-crew was 24. Aggression and emotional stability may be very defective below the age of 20, only to blossom later. Experience in other directions suggests that a not inconsiderable portion of youths under 20 showed psychiatric symptoms in the Forces which might suggest a poor prognosis, yet later examination and history have proved that their mental future seems relatively secure. (The reviewer is dismissing cases where the "illness" might suggest "malingering.")

The recognition that so much potential breakdown was satisfactorily coped with by unit station medical officers speaks much for the training of these officers in the simpler methods of dealing with mild mental symptoms. Such experience should be of encouragement to works' doctors in their cases of psychosomatic disorder, when the factors lie primarily in the working conditions.

Yet the industrial medical officer may well retort that in his constituency he deals not with selected air force personnel, but with a conglomeration of industrial units whose motives for employment are wholly different from those of air-crew. Something of the same sense of urgency may (we hope) be injected into the employee's outlook. It is clear that psychiatric screening practised in the Royal Air Force and the United States Army Air Corps yielded high dividends when the screening was combined with common-sense judgments, and such screening methods can be applied or experimented with in industry. They will expose fallacies as well as success; decisions to exclude certain workers from certain occupations will need to be combined with workable schemes for alternative occupations for the psychiatrically handicapped. Lastly, the happy experience of the Royal Air Force in relief employment (most conspicuously instructor duties between tours), hints at the desirability of relief employments in industry—an idea which runs counter to many present-day trade union conceptions.

Henry Wilson.

ABSTRACTS

(This section of the JOURNAL *is published in collaboration with the two abstracting Journals, Abstracts of World Medicine, and Abstracts of World Surgery, Obstetrics, and Gynæcology, published by the British Medical Association. The abstracts are divided into the following sections : toxicology ; industrial physiology ; industrial lung disease ; industrial dermatitis ; accidents and orthopædic surgery ; environment ; general.)*

TOXICOLOGY

A Case of Chronic Industrial Dinitrobenzol Poisoning. (Un caso di intossicazione professionale cronica da dinitrobenzolo.) CAPELLINI, A. and ZANOTTI, G. G. (1946). *Med. d. Lavoro*, 37, 265.

Dinitrobenzol is used in the preparation of colours and explosives, and may cause acute toxic symptoms. Cases of chronic poisoning are less common. In the case here described the patient worked in a factory and for 15 years had been making dinitrochlorobenzene. Into a boiler of 4 cubic metres capacity were put a cubic metre of water, a metric ton of pig iron, and hydrochloric acid. When effervescence occurred dinitrobenzol was added, and the boiler was then closed. The rise in pressure drove the mixture through an exhaust pipe into a filter, and then into the container, which received the dinitrochlorobenzene. Two exhaust tubes were placed near the boiler to trap any fumes, but, from a mechanical defect, the workman was exposed at intervals to powder and fumes of dinitrochlorobenzene. During the year before the patient came under treatment he noticed progressive asthenia, frontal headaches at work and later after work, some confusion, paræsthesiæ in the limbs, pruritus, insomnia, and loss of appetite. He also had some disturbance of vision. When he was first seen, there was a yellowish tint of the skin and sclerotics and a bluish coloration of the hands. On two occasions he spent a short time in hospital, each time being diagnosed as suffering from infective hepatitis. He then came under the authors' care. He was very weak, had disturbance of vision, paræsthesiæ in the limbs, and frequent fainting fits. His general condition was poor and there was some cyanosis of the extremities and of the lips, and gingival bleeding. No special physical signs were discovered. Blood examination revealed a secondary anæmia ; occult blood was found in the stools. After some weeks in hospital he gradually got better ; the headaches and paræsthesiæ lessened and the digestion improved. Vision did not improve much, and there were signs of optic neuritis. The cyanosis noted was undoubtedly due to methæmoglobinæmia, and there is no doubt that wrong diagnoses had been made on several occasions previously.

The icteric tint of the conjunctivæ can be associated with the positive Takata-Ara reaction. Though careful radiological examination was made, no sign of a gastro-intestinal lesion was noted, and it seems likely that scattered hæmorrhages were occurring in the gut as well as in the gums. The nervous and physical changes of confusional type and the changes in the optic nerve suggest direct poisoning of the central nervous system.

The authors stress the importance of protective clothing, of careful washing when work is finished, and of the particularly harmful action of alcohol, which favours methæmoglobin formation, according to Issakutz and Lipschitz. As absorption may also be through the respiratory tract, ventilation is important, as well as care in the design of the plant to avoid leakage of fumes.

G. C. Pether.

"Blue Haloes" in Atebrin Workers. MANN, I. (1947). *Brit. J. Ophthal.*, **31**, 40.

Six men were engaged in the production of atebrin tablets. They worked in an atmosphere which contained clouds of very fine atebrin dust for short periods. They had no dermatitis or other discomfort. Although protective clothing, goggles, and masks were worn, the fine dust penetrated these barriers and stained the skin of the hands, head, neck, and conjunctiva. After working for some weeks they noticed that at night a point source of light had a blue halo around it. One yard away the halo began 2 in. from the source and was about 3 in. wide. It was dark blue at the inner edge and pale blue at the outer. At a distance of 6 yd. faint yellow, green, and reddish-brown bands appeared outside the blue ring. All the men had normal visual acuity.

Slit-lamp examination showed yellow discoloration of the conjunctiva in the interpalpebral space, and a slight dulling and yellowing of the cornea which was barely visible macroscopically. Under magnification there appeared to be an actual deposit of minute dark-brown dots at the exposed margins of the limbus. The surface of the cornea was peppered with fine dust-like particles, which appeared dark yellowish-brown by direct illumination and opaque by transmitted light. The size of the particles was estimated as between 5 and 10 μ in diameter —that is, about the size of the nucleus of a corneal epithelial cell. The corneal surface was smooth and bright, and the change was present only in the surface layer of cells. In the lower part of each cornea there was a series of wavy yellow lines composed of closely aggregated dots similar to those peppered over the cornea but closer together and bright yellow. One patient noticed the haloes after being employed for less than a year, and they disappeared within 2 months of transfer to a different job. Later he returned to work with atebrin, and the phenomenon reappeared in 6 months although not reaching its previous intensity. There was no evidence of glaucoma in any patient, and it seems reasonable to attribute the condition to a diffraction effect.

Two volunteers, who had both taken 100 mg. of atebrin by mouth for 7 months, showed a pale-yellow staining of the skin generally, but their eyes were normal and they had never seen blue haloes. Atebrin dust

blown directly on to the eyes of rabbits produced in 6 days an appearance on slit-lamp examination similar to that of human subjects. These observations suggest that the corneal condition is caused by direct contamination with atebrin dust. The particles seen may not be atebrin itself, since this is soluble in the tears and the exact nature of the precipitates is doubtful, although they are probably an insoluble breakdown product of atebrin. Histological examination of an affected rabbit's eye was carried out, and the yellow granules in the cytoplasm could be seen in unfixed specimens. They were small and amorphous. The opaque yellow dots seen with the slit-lamp are the whole aggregation of these amorphous particles within the cell containing them. It was suggested that they might be composed of 2-chloro-7-methoxyacridone—an insoluble substance formed by hydrolysis of atebrin. *W. J. B. Riddell.*

The Estimation of Traces of Mercury in Urine. MILTON, R. F., and HOSKINS, J. L. (1947). *Analyst*, **72**, 6.

The method was developed for use in an investigation of mercury poisoning in a thermometer factory and a chemical factory. Colorimetric estimation with dithizone (diphenylthiocarbozone) is employed, the interfering effect of copper being eliminated by the use of extremely dilute dithizone in chloroform in the presence of 0·1 N hydrochloric acid. Organic matter is first removed by a permanganate-sulphuric oxidation and the mercury separated as sulphide, using arsenic as coprecipitant. The mixed sulphides are dissolved in aqua regia, and the nitric acid removed by hydroxylomine. Results for the 24-hour excretion of the factory workers and of normal persons are given. *H. M. Buckell.*

The Estimation of Mercury Compounds in the Atmosphere. MILTON, R. F., and DUFFIELD, W. D. (1947). *Analyst*, **72**, 11.

A brief description of the various methods of estimating mercury vapour in the air is given, none of which is accurate for mercury salts. Mercury vapour was trapped by alkaline hypobromite in three adsorption bubblers in series; the slip from the first bubbler was approximately 10%. Two bubblers were used in future. Mercury salts in the atmosphere were sampled, using a filter paper backed by a bubbler. The filter papers were treated in a similar manner to the organic matter handled by Milton and Hoskins (*Analyst*, 1947, **72**, 6). The hypobromite solution was acidified with hydrochloric acid, bromine removed with hydroxylamine, and the mercury estimated colorimetrically with dithizone in chloroform solution. Results of factory estimations are tabulated. *H. M. Buckell.*

The Biological Assay of Inhaled Substances by the Dosimetric Method. KAREL, L., and WESTON, R. E. (1947). *J. industr. Hyg.*, **29**, 23.

The physiological effects of substances which enter the body through the respiratory tract have usually been assessed in terms of the concentration to which the animals are exposed and the time of exposure in minutes. This method does not indicate the biologically active retained fraction of the inhaled substance; therefore the probable range of toxicity for man, in terms of animal experiments, has not been satisfactorily delimited. In the present investigation the lethal dose of phosgene was determined in goats, and also the percentage of inhaled agent retained: 62 normal unanæsthetized goats were employed, half of them being exposed to phosgene at a concentration of 1·19 mg. per litre, and the other half to one of 6·88 mg. per litre. The former group were exposed for 4·3 to 36·4 minutes, and the latter for 0·8 to 7·2 minutes. About a third of the animals survived, and it was found that there was no increase in the toxicity of the phosgene with increase of concentration. The median lethal dose on exposure to the lower concentration was 0·86 mg. per kilo of body weight, and for the higher concentration 0·84 mg. per kilo. The mean amount of retention for the 62 goats was 63%, while in previous observations on dogs it was found to be 74%. The goats showed marked respiratory inhibition on exposure to the phosgene, occasionally for as long as 60 to 97 seconds. This was longer than the time observed in dogs.

H. M. Vernon.

An Adaptation of the Dosimetric Method for Use in Smaller Animals. WESTON, R. E., and KAREL, L. (1947). *J. industr. Hyg.*, **29**, 29.

The median lethal dose and percentage retention of inhaled phosgene were determined in 37 normal unanæsthetized rhesus monkeys. The phosgene had a mean concentration of 1·6 mg. per litre, and the animals, being very susceptible, were exposed for only 0·4 to 3·1 minutes; 21 died 15 to 36 hours later, and the retained median lethal dose was found to be 0·2 mg. per kilo, or less than a fourth of that required for goats. The mean retention of inhaled phosgene was 79%. Respiration was to some extent affected, the mean respiratory rate under phosgene being 29 per minute as compared with 38 per minute in unexposed monkeys, while the average tidal air was 6·8 ml. per kilo as compared with 10·1 ml. per kilo. However, earlier investigators (Coon and others) found that low concentrations of phosgene (ranging from 0·45 to 0·8 mg. per litre) did not produce appreciable respiratory inhibition.

[The greater susceptibility of the monkeys as compared with the goats was due partly to their smaller size and relatively greater respiratory exchange. From the data recorded by the investigators it can be calculated that the relative respiratory exchange of the monkeys, which averaged 2·65 kg. in weight, was about 50% greater than that of the goats, which averaged 18 kg.]

H. M. Vernon.

Determination of Acetylene Tetrachloride in Air. GOLDENSON, J., and THOMAS, J. W. (1947). *J. industr. Hyg.*, **29**, 14.

Acetylene tetrachloride ($Cl_2HCCHCl_2$) is used in many industries because of its high solvent power and non-inflammability. It is one of the most toxic of halogenated aliphatic hydrocarbons, and it is, therefore, important to determine accurately the concentration of its vapour present in the atmosphere of industrial plants using the solvent. A concentration of 10 parts per million (0·068 mg. per litre at 25° C.) has been for some time regarded as safe for an 8-hour working day. The simple portable apparatus devised for the rapid estimation of low concentrations involved the use of a quartz combustion tube packed with platinum foil. The chlorides formed from the combustion of the vapours were absorbed in alkaline arsenite solution and determined volumetrically with 0·01N hydrochloric acid solutions. Laboratory and chamber tests proved that by this method 95% or more of the acetylene tetrachloride was recovered.

The degree of protection afforded by gas masks against the tetrachloride was determined by means of an elaborate apparatus. The maximum concentration of tetrachloride found in the air of the plant was 0·176 mg. per litre, but the gas canisters of the masks were tested at a concentration of 6 mg. per litre. Four different types of U.S. Army canisters were tested. They contained 250 to 640 ml. of impregnated charcoal, and were found to offer complete protection for at least 3 hours. A commercial type of canister, containing 1,150 ml. of ordinary activated charcoal, had a life of over 20 hours.

H. M. Vernon.

Fluorine Hazards. With Special Reference to Some Social Consequences of Industrial Processes. MURRAY, M. M., and WILSON, D. C. (1946). *Lancet*, **2**, 821.

In Lincolnshire lameness and cachexia occurred in cattle as a result of calcination of ironstone in the neighbourhood. The ironstone contained 1,200 parts per million (p.p.m.) of fluorine, which was reduced to about 300 p.p.m. on calcining with coal slack. The coal contained over 100 p.p.m. of fluorine. The smoke drifted on to vegetation and grass in the immediate vicinity, which showed values up to 2,200 p.p.m., and close to an adjacent farmhouse values of 68 to 487 p.p.m. Affected cattle excreted urine containing 26 to 69 p.p.m., and the fluorine content of their bones was up to 15,000 p.p.m. Water in the area contained only 0·5 p.p.m. People living in the farmhouse showed a moderate degree of dental fluorosis and excreted urine containing between 1·6 and 4·2 p.p.m. at a specific gravity of 1·015. These facts may be explained by surface contamination of food in the larder. The glass in the windows of the house facing the ironstone calcination process was etched. One inhabitant had suffered from stomach pains. On the farm wheat and barley embryos did not mature, and the farmer was paid compensation based on the difference between the expected and the actual yield of grain. In 6 years 7 horses and 11 cows had died, young cattle and sheep were under weight, the sheep were lame and had nasal discharge, and much poultry had been lost. At the height of calcining the density of fumes made driving on neighbouring roads difficult.

K. M. A. Perry.

Observations on the Effect of Diphenyl and Related Compounds upon Experimental Animals. DEICHMANN, W. B., KITZMILLER, K. V., DIERKER, M., and WITHERUP, S. (1947). *J. industr. Hyg.*, **29**, 1.

Diphenyl and its amino- and nitro-derivatives are constituents of various plastics and resins. As they are used commercially, their inhalation and skin contact cannot be avoided completely. Experiments were made on animals to determine tentative standards for safe industrial environments. The compounds, dissolved or suspended in olive oil, were administered by stomach tube to rabbits and by blunt hypodermic needle to rats. In the inhalation experiments a 160-litre chamber was employed, and the animals were exposed to the dust or vapour for 7 hours a day on 5 days a week.

The lethal doses per kilo of body weight (for 50% fatalities), when administered orally to rabbits, were 2·4 g. of diphenyl, 2·0 g. of *p*-nitrodiphenyl, 1·6 g. of *o*-nitrodiphenyl, 1·0 g. of *o*-aminodiphenyl, 0·7 g. of *p*-aminodiphenyl, and 0·2 g. of dihydroxyoctachlorodiphenyl, while the corresponding doses for rats were usually larger. These doses caused an increase of respiratory rate, lacrimation, muscular weakness, unsteadiness, and death in coma after 2 hours to 18 days. With one exception the repeated application of the compounds (in olive oil) to the abdominal skin of rabbits did not induce signs of local irritation, though sufficient absorption of the compounds took place to induce varying degrees of systemic poisoning. The exception was the dihydroxy compound above mentioned, and this induced marked signs of local irritation and systemic intoxication. Exposure of 6 rats for 46 periods to diphenyl dust at a concentration of 0·04 mg. per litre of air induced irritation of the nasal mucosa and 1 fatality, while one-fifth of this concentration caused no effects in rats but a few fatalities in mice. The absorption of all the compounds mentioned induced toxic myocarditis, hepatosis, nephrosis, and pneumonia, but the gastro-intestinal tract showed little if any evidence of local injury. Oral administration of sublethal doses of diphenyl or *o*-nitrodiphenyl to rabbits on 4 to 21 occasions caused no significant changes in the number of erythrocytes or leucocytes or in the concentration of hæmoglobin, but *p*-nitro- or *p*-amino-diphenyl induced anæmia together with hæmaturia or hæmoglobinuria. The *ortho*- and *para*-isomers of nitro- and amino-diphenyl are partially conjugated in the tissues of rabbits and are excreted as hexuronic acid and organic sulphates.

It was considered that, for prolonged human exposure, concentrations of 0·005 mg. diphenyl per litre of air should be regarded as dangerous, while concentrations of 0·003 mg. of *o*-nitrodiphenyl seem inadvisable.

H. M. Vernon.

The Acute and Chronic Toxicity of Methyl Chloride. I. SMITH, W. W., and VON OETTINGEN, W. F. (1947). *J. indlstr. Hyg.*, **29**,·47.

Various animals were exposed in chambers of 1,800 litres capacity. Methyl chloride was admitted through a rotameter, where it mixed with the air which was pumped through the chamber at a rate of 400 litres per minute. The animals were usually exposed to the vapour for 6 hours a day on 6 days a week. At a concentration of 4,000 parts per million (p.p.m.) the mean survival time of rats was 4 days, and of rabbits 13 days. At one of 2,000 p.p.m. it was 17 days for rats and 20 days for rabbits ; and at one of 1,000 p.p.m. it was 59 days for rabbits while two-thirds of the rats survived. Young animals usually withstood the methyl chloride better than adults ; thus, at 1,000 p.p.m. young mice survived 46 days and older mice only 11. At the low concentration of 500 p.p.m. dogs and monkeys showed a high degree of susceptibility. Of the 4 dogs exposed, 3 died after 2 to 4 weeks' exposure, while the 2 monkeys exposed died after 16 and 17 weeks. If it is assumed that the susceptibility of human beings is comparable to that of dogs and monkeys, it follows that daily exposures to concentrations of 500 p.p.m. are extremely dangerous even for a period of 2 weeks or less. The maximum allowable concentration for exposures of relatively long duration must be well below 500 p.p.m.

H. M. Vernon.

The Inhalation Toxicity of Ketene and of Ketene Dimer. WOOSTER, H. A., LUSHBAUGH, C. C., and REDEMANN, C. E. (1947). *J. industr. Hyg.*, **29**, 56.

Ketene ($CH_2 : CO$) is used as an acetylating agent in the laboratory and in industrial plants. Animals were exposed in a 40-litre chamber, through which air was drawn at a rate of 50 to 100 litres per minute. Ten-minute exposures were made, and the following samples of the results obtained may be quoted. At a concentration of 0·12 mg. per litre 29 out of 40 mice died in 115 to 240 minutes ; at one of 0·43 mg. per litre all the 4 rats

exposed died in 150 minutes or less ; at a concentration of 0·63 mg. per litre 5 out of 8 guinea-pigs died in 8 hours to 3 days. The few tests made with ketene dimer (diketene) showed that it is much less toxic than ketene. For instance, only 1 mouse out of 20 died at a concentration of 3 mg. per litre. After death the animals appeared normal, but pressure on the thorax caused œdema fluid to flow profusely from nose and mouth. The lungs were hyperæmic and mottled by lobules filled with œdema fluid. It is concluded that ketene is as toxic as phosgene and is comparable to it in its action. *H. M. Vernon.*

Does Welder's Disease Really Exist? (Existe-t-il une maladie des soudeurs ?) FEIL, A. (1946). *Presse méd.*, **54**, 598.

Arc welders' methods of working are described. The fumes from the electrode metal and its covering did not contain toxic substances. An inquiry was started on 67 arc welders, most of whom had been working for at least 5 years, and some for 15 or 20 years. No serious intoxications or pathological manifestations were found. Frequently welders suffer from the condition of "flash-eye" (*coup d'arc*) from exposure to ultra-violet and infra-red radiations, with characteristic symptoms and a delayed and transient kerato-conjunctivitis. Weakness of vision, especially at night, was found in 20%. Digestive disturbances occurred in 37%. Cough is fairly common (37%), and in radiographs of 1 in 10 or 12 workers there is an appearance of nodulation (*granitée*), which is due to iron oxide, since the dust contains 51% of iron oxide and only 1·5% of silicon dioxide. These small nodules at necropsy do not show the typical connective-tissue proliferation of silicosis. There was little evidence of abnormalities in the blood or in the nervous system. The evidence that welding causes sexual dysfunction was slender, for only 4 to 5% complained of impotence, and this could be attributed to other causes. Intoxication from lead, phosphorus, and arsenic is possible, but only because of contact with these substances and not specifically because of welding. The ocular manifestations alone seem to be directly caused by the occupation. The risks of welding have been over-emphasized. *J. N. Agate.*

Korsakow's Syndrome in Relation to Atrophy of the Brain and Carbon Monoxide Poisoning. (Zespól korsakowa w zwiazku z zanikiem mózgu i wodoglowiem po zatruciu tlenkiem wegla. Studium klinicznorentgenologiczne). BILIKIEWICZ, T. (1946). *Polsk. Tyg. lek.*, **1**, 1256.

Mental disturbances due to carbon monoxide poisoning assume in most cases the clinical form of Korsakow's syndrome, memory defects, compensatory fantasies, and lowering of the intelligence standard being the main features. The disturbances are often preceded by an interval of apparent normality lasting for from a few hours to several weeks.

In 1923, a man, aged 30, suffered severe carbon monoxide poisoning and remained in coma for 5 days. Two weeks later he was able to return to work, but 3 weeks after this apparent recovery he was admitted to hospital with grave neuromotor and mental symptoms : muscular rigidity, exaggeration of all tendon reflexes, ankle clonus, bilateral Babinski sign, abolition of the superficial abdominal reflexes, and incontinence of the sphincters ; mentally there was dullness, apathy, and loss of memory. He remained in hospital for 5 months, by which time the motor signs had subsided and he had recovered the use of his limbs ; his intelligence improved, although it was still impaired when he was discharged. In 1944—21 years later—he was re-admitted to the same hospital. Only slight traces of neuromotor disturbances were found—such as drooping of the left angle of his mouth and some exaggeration of his ankle jerks, particularly of the left one—but there was an unmistakable Korsakow's syndrome, with its classical symptomatology. No evidence of syphilis in blood or cerebrospinal fluid was found. Encephalography showed a uniform enlargement of all the cerebral ventricles. The radiological diagnosis was atrophy of the brain and internal hydrocephalus. *A. Lilker.*

Estimation of Sulfur Compounds in Air. FEINER, B., BURKE, W. J., and MOSKOWITZ, S. (1946). *J. industr. Hyg.*, **28**, 276.

To determine the atmospheric concentrations of onion-oil vapour (largely allyl propyl disulphide) and of xylyl mercaptan, the apparatus previously used for the estimation of halogenated hydrocarbons in air was employed. Air at the rate of 1 litre per minute is drawn through a quartz combustion tube containing a platinum catalyst, and heated to about 900° C.; then through a gas absorber containing glass beads wetted with a 2% solution of sodium carbonate. The sulphur of the sulphide is converted into sulphur dioxide, which in turn forms sodium sulphite. One-hour samples were taken in the breathing zone of the workers and were analysed by titration with standard barium chloride solution, using tetrahydroxyquinone as an internal indicator for excess barium ion. The titration is made sensitive by the use of ethyl alcohol to decrease the solubility of the barium sulphate formed. The method can be used when the quantity of sulphur in the air sample is 0·1 mg. or more. *H. M. Vernon.*

Fever among Foundry Workers. (La fievre des fondeurs et sa pathogenie.) FEIL, A. (1946). *Med. Usine.* **8**, 254.

The author studied this symptom in several workers in a soldering workshop. He reports 8 cases occurring in his own practice. He believes that the fever is due to zinc, although it cannot be regarded as a direct acute poisoning from this metal, and it would be more correct to consider it the result of a pathogenetic effect of zinc oxide on the pulmonary epithelioma. The proteins of the blood plasma, under the caustic action of zinc vapours, are apt to form heteroproteins, the passage of which into the blood stream would, perhaps, explain the fever. *L. Roche.*

Percutaneous Absorption of Carbon Bisulphide. (Sull' assorbimento percutaneo del sulfuro di carbonio.) NUNZIANTE CESARO, A. (1947). *Med. d. Lavoro*, **38**, 54.

Two reasons for suggesting that carbon bisulphide may enter the body through the unbroken skin are, first, that the substance is soluble in fats and could thus pass through, and secondly that, as the heavy vapour lies near the ground, absorption through the feet and legs is favoured, which would explain the greater frequency of polyneuritis in these parts. The author rejects the first assumption and does not consider that there is adequate proof that any substance absorbed by the skin must necessarily be most harmful to the tissues closest to the area of penetration. The nerves of the legs are the longest in the body. Furthermore, fatigue is most often felt in the legs. Many toxic substances which are not absorbed through the skin tend to attack the nerves of the legs.

Ten men were shut in a closed chamber except for their

faces. Fresh air was breathed from a mask. One arm was encased in cotton-wool soaked in carbon bisulphide, and on the floor of the chamber were placed pieces of cotton-wool soaked in the solution. The expired air was collected in a closed circuit from the mask and tested for carbon bisulphide. Subjects were exposed to the gas for 20 minutes. In none of the ten subjects tested was any of the gas detected in the expired air, and the author concludes that neither liquid nor gaseous carbon bisulphide is absorbed by the unbroken skin. *G. C. Pether.*

The Artificial Fibre Industry and its Dangers. (L'industrie des fibres artificielles et ses dangers.) AUFFERT, J. (1946). *Arch. Mal. Prof.*, **7**, 181.

Conditions in the artificial fibre industry, the technical methods employed, and the risks run by the workers, are described. The main risk is carbon disulphide poisoning due to the "viscous" process. In the artificial fibre industry in France approximately 3,000 persons are exposed to carbon disulphide poisoning. In 1942 there were 252 cases of poisoning, 69 of them serious. By 1945, there were only 62 cases, all mild. The measures taken to combat the hazard are as follows :

1. *Medical.*—Careful selection of personnel ; regular analysis of the atmosphere, this being especially frequent where high concentrations are found (examination every four or six weeks in exposed places has considerably reduced the number of grave cases of poisoning).

2. *Technical.*—Individual precautions such as the wearing of a gas-mask (though this hampers the worker) ; and collective precautions such as purifying the atmosphere in the workshops and psychological education of the workers. *L. Roche.*

Arsine Poisoning, Mode of Action and Treatment. KENSLER, C. J., ABELS, J. C., and RHOADS, C. P. (1946). *J. Pharmacol.*, **88**, 99.

Following the work of Peters, which showed that a dithiol protected against lewisite, a related substance has been found of value in arsine poisoning. This paper summarizes results of *in vitro* and *in vivo* experiments in which 2,3-dimercaptopropyl-ethyl-ether was found effective if given soon enough after exposure to arsine. Other protective substances were investigated. Arsine was generated from magnesium arsenide and water, the concentrations being estimated by a modification of the molybdenum-blue method. *In vitro* experiments with erythrocytes were carried out by adding 0·4 ml. of serum to 1·6 ml. of defibrinated blood. The cells were exposed to 0·2% arsine at 37·5° C. for 2 hours. Protective substances were added 5 minutes after exposure started. Lysis was determined colorimetrically. Inhibition of tissue respiration was also measured in the Warburg apparatus, in which tissue respiration of control material remained reasonably constant for 3 hours.

The average percentage lysis produced by arsine for 150 experiments was 33·3%. The variation in response of cells from one donor was small as compared with responses from different donors : with 2,3-dimercaptopropyl-ethyl-ether, 4·8% lysis ; with the product "BAL," effective against lewisite, 17% lysis occurred. Good protection was obtained with ascorbic acid and sodium thiosulphate in high concentrations, though at the level of concentration of the thiols in these experiments they are only 10% as effective. Of the compounds giving good protection against lysis of erythrocytes, only the dithiols gave protection to tissue-slice respiration *in vitro* ; 2,3-dimercaptopropyl-ethyl-ether gave almost complete protection of kidney and brain and some protection of liver. BAL gave good protection of kidney and brain, but accelerated inhibition by arsine of liver-slice respiration. BAL was found to give a small but inconstant protection to rabbits, dogs, and monkeys exposed to arsine. In rabbits arsine had the effect of increasing the toxicity of BAL, and thus of reducing the protective action. The authors point out that in lewisite-poisoning several lethal doses of BAL may be administered to neutralize lethal doses of lewisite.

Other compounds were found to be more potent and were given in 3 intramuscular injections of successively smaller doses. The second was given 4 hours after the first, and the third 24 hours later. Using the more effective compounds, particularly 2,3-dimercaptopropyl-ethyl-ether, almost complete protection was afforded if treatment was started 30 minutes after exposure. Survival was reduced to 90% at 1 hour, 40% at 2, 3, and 4 hours, and 20% at 5½ hours. 1,6-dimercaptohexane was almost as good if given within 1 hour. If used with BAL a 50% survival was maintained between 2 and 4 hours—higher than was obtained with either separately. The percutaneous route of administration was also used, being preferred to the intravenous route, which had been reported by Cattell as responsible for damage to the central nervous system in cats. No such effects were observed after intramuscular or percutaneous administration. Local application to human skin produces no irritation, and other workers have applied 2,3-dimercaptopropyl-ethyl-ether percutaneously to man in doses of 50 mg. per kilo without ill effect. *R. H. D. Short.*

Clinical Uses of 2,3-Dimercaptopropanol (BAL). I. The Systemic Treatment of Experimental Arsenic Poisoning (Mapharsen, Lewisite, Phenyl Arsenoxide) with BAL. EAGLE, H., MAGNUSON, H. J., and FLEISCHMAN, R. (1946). *J. clin. Invest.*, **25**, 451.

"BAL" (British anti-lewisite) injected into rabbits by the subcutaneous, intramuscular, or intravenous route in aqueous or propylene glycol solution is effective in the treatment of acute and subacute mapharsen poisoning. A more stable BAL preparation is obtained by dissolving it in peanut oil with the aid of benzyl benzoate (2 g. for each g. of BAL). This preparation can be sterilized by heat with only slight loss in activity and is suitable for human use. It also has proved effective in the treatment of mapharsen, lewisite, and phenyl arsenoxide poisoning in rabbits. BAL exerts an antidotal action through its ability to remove arsenic from its combination with cells, with the excretion of the stable and relatively non-toxic thioarsenite so formed. The rate of urinary arsenic excretion is strikingly increased, in some cases by more than a hundredfold.

G. R. Cameron.

Clinical Uses of 2,3-Dimercaptopropanol (Bal). II. The Effect of BAL on the Excretion of Arsenic in Normal Subjects and After Minimal Exposure to Arsenical Smoke. WEXLER, J., EAGLE, H., TATUM, H. J., MAGNUSON, H. J., and WATSON, E. B. (1946). *J. clin. Invest.*, **25**, 467.

Twelve men were exposed for 6 minutes to very low concentrations of an arsenical smoke (diphenylcyanoarsine) and their urinary excretion of arsenic estimated. There was an immediate increase in the urinary arsenic with a gradual fall from an initial average of 6·8 μg. per hour to an average of 3 μg. per hour 72 hours after exposure. The corresponding value in a group of 6 control subjects was 2 μg. per hour. Both groups were given a single intramuscular injection of "BAL" (3·5 mg.

per kilo) in the form of a 10% solution in peanut oil and benzyl benzoate. A significant increase in the rate of urinary excretion resulted, reaching a maximum within 2 to 4 hours, and most pronounced in the subjects exposed to the arsenical smoke. Since this effect on excretion did not persist for more than 4 hours, it is concluded that such an interval is the optimum between successive therapeutic injections of BAL.

G. R. Cameron.

Clinical Uses of 2,3-Dimercaptopropanol (BAL). III. Studies on the Toxicity of BAL on Percutaneous and Parenteral Administration. SULZBERGER, M. B., BAER, R. L., and KANOF, A. (1946). *J. clin. Invest.*, **25**, 474.

The toxic effects of skin application or intramuscular injection of "BAL" to healthy volunteers is discussed. Approximately 0·3 g. BAL applied to the skin in the form of BAL 10% in K–Y jelly or BAL 10% in petrolatum gave no unusual effects in 5 males except for transitory local erythema, whealing, and itching at the site of application. Approximately 1·7 g. of BAL applied to large areas of the skin in the form of a 5% concentration in K–Y jelly produced no convulsive phenomena, blood changes, kidney damage, or other forms of systemic poisoning in any of 5 healthy males. With intramuscular injection, toxic manifestations appeared at a dose of 3·6 to 5·5 mg. per kilo body weight, and included nausea, vomiting, headache, burning sensation of lips, mouth, and throat, feeling of constriction and sometimes pain in throat and chest, burning and tingling of extremities, conjunctivitis, lacrimation, rhinorrhœa, salivation, sweating of forehead and hands, abdominal pain, and general agitation. Local pain at the site of injection and muscle spasm of the leg were also common. An elevation of the systolic and diastolic blood pressures occurred within half an hour of the injection. When the first two doses were given with an interval of only 2 hours cumulative toxic effects were observed, but 4 doses each of about 5 mg. per kilo at 4-hour intervals produced no severe or lasting damage in the average normal individual.

G. R. Cameron.

Clinical Uses of 2,3-Dimercaptopropanol (BAL). V. Skin Sensitization to BAL. SULZBERGER, M. B., BAER, R. L., and KANOF, A. (1946). *J. clin. Invest.*, **25**, 488.

Small amounts of "BAL" preparations were rubbed into the forearm of human volunteers on 14 consecutive days. When applied in the form of 5% BAL in grease ointment and 5% BAL in carbowax ointment and 5% BAL in ethylene glycol, all gave sensitization dermatitis in 16 out of 88 subjects (19% of exposed subjects). Application to damaged skin gave sensitization in 35 of 53 subjects (63%). Sensitization reactions included erythema, œdema, and papules, the exposed area being more strongly sensitive than other parts of the body. Repeated intramuscular injection of BAL also produced skin sensitization as demonstrated by positive patch tests in 5 of 18 subjects. *G. R. Cameron.*

Clinical Uses of 2,3-Dimercaptopropanol (BAL). VII. The Treatment of Arsenical Dermatitis with Preparations of BAL. LONGCOPE, W. T., LUETSCHER, J. A., WINTROBE, M. M., and JAGER, V. (1946). *J. clin. Invest.*, **25**, 528.

Seven patients with an intractable localized dermatitis caused by diphenylamine chlorarsine improved within a few days after the inunction of "BAL" (1 to 10%) ointment, starting with daily applications of 100 mg. and increasing to 500 mg., Fifteen cases of generalized exfoliative dermatitis following the use of antisyphilitic arsenicals showed symptomatic and objective improvement after inunction or intramuscular administration of BAL. The duration of the dermatitis in over half the patients so treated was shorter than in a comparable group not treated with BAL. A mild recurrence of the dermatitis was frequent when treatment was not continued for at least a week, but 6 such relapses cleared quickly when therapy was re-instituted. BAL ointment was quite painful when applied to inflamed skin, but intramuscular injections were much less disturbing.

G. R. Cameron.

Clinical Uses of 2,3-Dimercaptopropanol (BAL). VIII. The Effect of BAL on the Excretion of Arsenic in Arsenical Intoxication. LUETSCHER, J. A., EAGLE, H., and LONGCOPE, W. T. (1946). *J. clin. Invest.*, **25**, 534.

Further support is given for the contention that "BAL" increases urinary excretion of arsenic. Of 24 courses of BAL to 16 patients with arsenical intoxication and 2 patients receiving arsenical treatment without reaction, 16 were followed by a definite increase in arsenic excretion, 4 by a possible increase, and 4 by no increase. The increase appears to be more consistent in patients with the arsenical dermatitis than in hepatitis, suggesting a correlation with the greater efficacy of BAL in the treatment of arsenical dermatitis.

G. R. Cameron.

Clinical Uses of 2,3-Dimercaptopropanol (BAL). IX. The Treatment of Lewisite Burns of the Eye with BAL. HUGHES, W. F. (1946). *J. clin. Invest.*, **25**, 541.

A single instillation of 5% "BAL" solution or ointment within 2 to 5 minutes after exposure to lewisite droplets or vapour effectively prevents the development of serious ocular lesions in the rabbit. BAL penetrates the cornea rapidly and withdraws toxic arsenical material from the tissues before irreversible changes have developed. *G. R. Cameron.*

Clinical Uses of 2,3-Dimercaptopropanol (BAL). X. The Treatment of Acute Systemic Mercury Poisoning in Experimental Animals with BAL, Thiosorbitol and BAL Glucoside. GILMAN, A., ALLEN, R. P., PHILIPS, F. S., and ST. JOHN, E. (1946). *J. clin. Invest.*, **25**, 549.

This is an experimental study of the value of "BAL," BAL glucoside and 1-thiosorbitol in the treatment of acute mercurial poisoning in rabbits and dogs. Rabbits receiving 3 mg. of $HgCl_2$ per kilo—a dose giving 96% of deaths—intravenously were completely protected by 3 doses of 0·1 mM. per kilo of any of the above compounds, provided therapy was initiated within 5 minutes. BAL glucoside was the most potent, 1-thiosorbitol the least. Dogs given 4 mg. of $HgCl_2$ per kilo intravenously—a fatal dose—were completely protected from the renal effects of the metal by 3 equal doses of BAL totalling 0·15 mM. per kilo when treatment was delayed for 30 minutes. Even when treatment with BAL glucoside or BAL was delayed for 2 hours protection was striking. Of 44 dogs receiving 30 mg. of $HgCl_2$ per kilo orally—a fatal dose—in which treatment with a total dose of 0·15 mM. of BAL or BAL glucoside per kilo was delayed for 2 to 5 hours, 28 survived, and only 1 animal died in uræmia. *G. R. Cameron.*

Clinical Uses of 2,3-Dimercaptopropanol (BAL). XI. The Treatment of Acute Mercury Poisoning by BAL. LONGCOPE, W. T., and LUETSCHER, J. A. (1946). *J. clin. Invest.*, **25**, 557.

This paper reports the successful treatment of acute mercurial poisoning in humans with intramuscular injections of "BAL." Eight patients swallowed not more than 0·5 g. of mercury bichloride. Treatment with BAL was started from 20 minutes to 3½ hours later. All recovered promptly. Six patients swallowed 1 g. Five were treated within 1 to 3½ hours, and all recovered within 2 to 8 days. One patient who was treated initially with small amounts of BAL 13 hours after taking 1 g. of mercury bichloride died on the ninth hospital day. Nine patients took from 1·5 to 20 g. of mercury bichloride, 5 of the 9 having swallowed more than 1·5 g. Eight were treated with BAL from 1½ to 3½ hours after taking the poison, and one was first treated 19 hours after having swallowed at least 1·5 g. This patient was well in 3 weeks, and the other 8 in 2½ to 7 days. The initial amount of BAL used for the first intramuscular injection in 21 patients was 300 mg. (3 ml. of 10% solution). Two patients, including the one who died, received an initial dose of only 150 mg. Twenty-one patients received from 450 to 750 mg. in the first 12 hours, and a total of 900 to 2,870 mg. in a period of 3 to 4 days. Toxic features attributable to BAL were observed in a few patients after the intramuscular injections of 300 mg. or, in some instances, of 150 mg. of BAL. The authors emphasize prompt treatment by BAL in an initial intramuscular injection of 300 mg., followed within the first 12 hours by 2 or even 3 further injections of 150 mg. each.

G. R. Cameron.

Tests of 2, 3-Dimercaptopropanol and Related Dithiols for Decontamination of Lewisite on Human Skin. THOMSON, J. F., SAVIT, J., and GOLDWASSER, E. (1947). *J. Pharmacol.*, **89**, 1.

Lewisite was applied to the skin of human volunteers in amounts sufficient to produce erythema and vesication. A micropipette delivering multiples of 0·05 c.mm. lewisite was employed. The contaminated area was treated with the new dithiol after 40 minutes. Adequate controls using BAL or leaving the lewisite area untreated were instituted. The authors agree with other investigators that the vesicant action of small amounts of lewisite can be prevented by BAL, and they believe that BAL is still the most effective decontaminant against lewisite. They show that effectiveness is dependent largely upon (1) the dose of lewisite used, (2) the interval between contamination and decontamination, (3) the concentration of the dithiol in the vehicle, and especially (4) the temperature and relative humidity of the immediate environment of the test subject. Water-soluble derivatives of BAL have advantages, since they are less toxic and have a less objectionable odour, but they are not so effective as BAL. BAL retains its pre-eminence as a treatment for lewisite, not only because of its superior local effects, but also because it is effective against systemic effects and is a potent agent in the treatment of eye damage by lewisite.

G. R. Cameron.

The Effect of BAL on the Excretion of Arsenoxides. CHANCE, A. C., and LEVVY, G. A. (1947). *Quart. J. exp. Physiol.*, **34**, 79.

The subcutaneous administration of BAL (30 mg. per kilo of body weight of a 5% solution in peanut oil) was found to increase the rate of excretion of arsenic in the urine of rabbits to which arsenoxides (phenylarsenoxide and mapharsen) had been administered intravenously in doses of 0·42 to 3·4 mg. of arsenic per kilo of body weight. The rate of excretion of arsenic in the fæces was not altered.

R. H. D. Short.

INDUSTRIAL PHYSIOLOGY

Basovascular Spasm. GURDJIAN, E. S. (1946). *Industr. Med.*, **15**, 481.

Pathological effects occur with pneumatic tools working at an intensity of 12,000 or more vibrations per minute. Three types of disease have been described; the type affecting bones and joints; the neuritic type; and the vasopastic type. Combinations may exist of any of these. The elbow- and shoulder-joints are those most frequently involved, and there seems at times to be a degeneration of the small bones of the hand. Tenosynovitis may occur. The peripheral nerves most involved are the median, ulnar, and radial. The incidence varies from 30 to 80%. Exposure to cold causes whiteness, numbness, and pain, mostly in the left hand. Warm air changes the colour and causes severe pain. Biopsies have shown normal vessels, with no evidence of Buerger's or Raynaud's disease. The mechanism of the condition is not known. The hand involved gets the most vibration; and the position of the tool may be significant. Cold air is an important factor. There seems to be an alteration in the chemistry of the tissues.

Treatment is mostly preventive, usually reduction of exposure. Mechanical means of holding tools have also been devised, and other tools have been substituted at times. Care should be maintained in the selection of employees for this intermittent operation. Massage and other modalities are not effective. Sympathetic block has been tried with some promise. [From the official report.]

Studies on Altitude Decompression Sickness. I. Symptomatology. GRAY, J. S., MAHADY, S. C. F., MASLAND, R. L., and WIGODSKY, H. S. (1946). *J. Aviat. Med.*, **17**, 333.

This paper deals with investigations at the U.S. Army Air Force School of Aviation Medicine during 1942 and 1943, and catalogues the various types of symptoms experienced in the decompression chamber during 2,920 man-flights of 2 to 4 hours at simulated altitudes of 35,000 to 38,000 ft. (10,500 to 11,500 m.) while breathing 100% oxygen. The frequency of the various symptoms of decompression sickness is given, also their degrees of intensity and duration, the time interval at 38,000 ft. before they occurred, and the altitude at which relief was experienced on descending. The subjects of these investigations were chiefly flying cadets and pilots aged between 18 and 30 years.

The commonest symptoms necessitating forced descent were bends in 73% of cases, and chokes in 17%. A classification of the symptoms present at the time of descent is given.

Gut distension causing colicky pain occurred in the majority of subjects, usually in the first half-hour at 35,000 to 38,000 ft., and was generally of a transitory nature passing off in 60% of the cases without any descent. Where descent was necessary it had to be made on an average within 11 minutes of the beginning of pain, but only in about 4% of the total subjects tested was it essential to descend to the ground owing to abdominal colic. Circulatory disturbances of the nature of shock accompanied 26% of the cases of bends

or chokes, the great majority being of the secondary type. Symptoms usually took several hours to disappear after return to ground and rest. Neurological symptoms were rare and chiefly affected the vision—scotomata; they usually cleared up within an hour or so of descent. Skin rashes and œdema of the soft tissues of chest and abdominal wall were rare and occurred in about 0·6% of the total personnel tested. At times a profuse flow of mucus from the head sinuses accompanied the expansion of gases at higher altitudes, and so interfered with breathing as to necessitate descent. Toothache so severe as to warrant descent occurred in about 0·34% of the subjects tested.

[Chokes or substernal distress with burning sensation, lung cough made worse by deep breathing, dyspnœa, and apprehension appear to be far commoner in the U.S.A. than in the U.K., where they are rarely seen; whether this is a climatic or racial peculiarity is undetermined.] *H. E. Whittingham.*

Formulation of the Principal Factors Affecting the Rate of Uptake of Carbon Monoxide by Man. PACE, N., CONSOLAZIO, W. V., WHITE, W. A., and BEHNKE, A. R. (1946). *Amer. J. Physiol.*, **147**, 352.

The rate of uptake of carbon monoxide has been shown to be constant with respect to blood concentration of COH*b* [carboxyhæmoglobin], up to values of one-third the equilibrium level, when air containing CO in the range 1 part to 20 parts per 10,000 is breathed by men at rest or engaged in moderate physical activity. An equation has been derived which serves as a means of estimating in man the degree of blood saturation with CO as a result of exposure to air containing this gas as follows:

$$\Delta \text{ per cent. COH}b = \frac{\text{Parts CO} \times \text{minute volume} \times \text{exposure time.}}{46{\cdot}5 \times \text{blood volume}}$$

The equation is valid for values of per cent. COH*b* up to one-third the equilibrium value for the air concentration of CO under consideration. Within this range Δ per cent. COH*b* may be estimated within a degree of error whose standard deviation is $\pm 2{\cdot}3\%$ COH*b*.

The fraction of CO removed from the inspired air by the blood was found to be constant as uptake progressed, and the mean for a group of 12 men was 41·2% with a standard deviation of $\pm 4{\cdot}4\%$. This value is somewhat lower than that observed by previous investigators. —[From the authors' summary.]

Inflammation of the Palmar Aponeurosis in P.T.T. (Post Office) Fitters. (L'aponévrosite palmaire chez les ouvriers-monteurs des P.T.T.) DESPLAS, M. B. (1946). *Arch. Mal. prof.*, **7**, 217.

In 21 years the author saw over 200 cases of Dupuytren's contracture, and he recently observed a group of workers exhibiting a high incidence. One, a vigorous man of 48, had a palmar contracture affecting the fourth and fifth fingers of the right hand. The left hand was normal. This workman was required to lay electric wires of varying diameters, stripping these of the protective lead, paper, or pitch when it was necessary to cut them. A considerable muscular effort was needed to close the scissors provided, which had a leverage of 1 : 1, and when the author attempted to do the work he was unable to cut the thicker wires. He observes that a strong man can exert a gripping force of 50 kg. as measured with the dynamometer in the hand. To cut some of the wires a force of 75 kg. was needed, and this fell chiefly on the palmar fascia leading to the fourth and fifth fingers, as they worked against the thenar side of the palm. He did not exclude the possibility of some specific irritation of the palm by the pitch used as an insulator. The incidence of palmar contracture in cable workers was very high. It generally appeared about the age of 34 to 40 after from 3 to 7 years' employment. In one area 9 out of 32 employees exhibited the lesion, and these were mostly men working on thick wires. The author feels that the badly-designed and blunt scissors now in use should be replaced by a new pattern giving a leverage of 1 : 3 or 1 : 4. *G. C. Pether.*

Some Observations on Men Sitting Quietly in Extreme Cold. HORVATH, S. M., GOLDEN, H., and WAGER, J. (1946). *J. clin. Invest.*, **25**, 709.

Forty-five soldiers (average age 20·5 years; surface area 1·79 sq. metres) in excellent physical condition served as subjects. Each submitted to 2 to 5 tests in a particular environment after a light breakfast 2 to 3 hours before entering the cold room. Half-an-hour before exposure the men donned thermocouple harnesses and their underwear and lay quietly in the control room at 22° C., 50% humidity, for the measurement of basal skin and rectal temperatures. This done, they dressed in their outer clothing and sat quietly in the particular environment for 3 hours. At 22° C. the metabolic rate increased by approximately 5%. Exposure to cold environments at temperatures of 1·1°, −17·8°, −23·3°, −26·1°, −28·9°, −34·4°, and −40·0° C. was accompanied by increased metabolic rate, which was greatest in the third hour and least in the first. Although no correlation between environmental temperature and metabolic response was noted, at −40° C. the metabolic rate averaged 74% of the basal value in the third hour. Mean skin temperatures calculated from five areas (thigh, toe, arm, calf, and chest) fell particularly in the first hour, the rate decreasing in the third hour. Rectal temperatures fell during the first hour but remained relatively constant in the final 2 hours except at −26·1° and −34·4° C.; the colder the ambient air the steeper the fall observed in skin temperature. Isolated observations are of little value, as an individual may begin with a high initial value but end the period of exposure with one of the lowest values recorded in a group of individuals examined simultaneously. At −28·9° C., less than 10% of the subjects shivered in the first hour, 50% shivered before 120 minutes, and 75% under 151 minutes. The greatest temperature changes were exhibited in the hands and feet, toe temperatures below 0° C. being encountered at −23·3° to −40·0° C. *C. C. N. Vass.*

The Reactions of Tissue to Cold. The Pathology of Frostbite, High Altitude Frostbite, Trench Foot, and Immersion Foot. FRIEDMAN, N. B. (1946). *Amer. J. clin. Path.*, **16**, 634.

The local tissue injury in the extremities caused by various forms of exposure to cold has been studied in more than 100 cases. Material has not been examined during exposure or during the pre-hyperæmic vasoconstrictive stage. In the hyperæmic œdematous stage the vessels became dilated and engorged with red cells and the tissues filled with fluid. Thrombi then formed from the agglutinated erythrocytes, and secondary ischæmic reactions followed. The skin showed vesiculation; the sweat glands and ducts vacuolation, degeneration, atrophy, cystic dilatation, and squamous metaplasia; and the nerves degeneration of the axis cylinders, demyelination, lipoid phagocytosis, and later scarring.

Atrophy, degeneration, and necrosis were widespread in the muscles. The connective-tissue fat became crystallized and necrotic, lipoid phagocytes and oil cysts appeared, and free and intracellular fat was seen in the vessels. The adipose tissue (presumably at a later stage ; ? during repair) showed atrophy and scarring, an interstitial sclerosis similar to that seen in reaction after irradiation. Non-sequestrating necrosis and atrophy of bone also occurred with the formation of new bony lamellæ apposed to the dead trabeculæ. Most of the changes in both superficial and deeper structures are attributed to the vascular occlusion resulting from thrombosis, and it is believed that injuries resulting from exposure to cold under a variety of conditions have a similar pathogenesis in which thrombosis plays the most important part. Structures rich in fats and lipids (adipose tissue and myelinated nerve fibres), however, appear to be affected directly by cold. *G. Popják.*

Control of Heat in Industry. MACHLE, W. (1946) *Occup. Med.*, **2**, 350.

The author first describes the concept of thermal equilibrium, according to which the mean temperature of the body when exposed to heat rises to a new level, which may be as high as 103° F. An upper limit of 90° F. wet-bulb temperature for saturated air is about the highest which can be tolerated, levels 2° to 3° F. lower being necessary for clothed men. Some authorities suggest a rise of 1·5° F. of rectal temperature as the permissible maximum ; but men have been observed to work for 4 hours or more with rectal temperatures of 103° F. It has been noted that high radiation causes a marked increase in metabolism. When general ventilation and cooling are impossible local cooling in suits and jackets or by localized air currents may be useful.

After a period of adaptation persons selected for work involving exposure to heat should work for 4 or 5 days in a hot atmosphere to the limit of tolerance. The greatest degree of toleration is seen in men between the ages of 21 and 28, after which there is a steep fall. Attempts should be made to lessen heat production in persons exposed to heat. As little output of work as 92 kg.-metres per minute is equivalent in heat production to a rise in air temperature of 12·5° F. After men leave hot environments the drop in body temperature seems to follow Newton's law, the greatest loss being in the early periods of exposure. Thus it would appear that frequent short intervals in cooler air will give more relief than rare and longer rest pauses. Men working at a wet-bulb temperature of 90° F. showed a drop in rectal temperature from figures between 100° and 104° F. to a point only 0·2° to 0·4° F. above normal. The degree of relief given may be estimated to some extent from the pulse rate, which should be between 125 to 130 for fit young men. The loss of 1·5% of body weight by water deficit is equivalent to a rise in air temperature of 12° F. or an increase in work output of 92 kg.-metres per minute. The body may lose in 1 hour an amount of water equal to the blood volume, yet deterioration is seen with a loss as low as 6% of body weight. Thirst is not an adequate measure of water deficiency, and the author notes the need for salting the water to the extent of 0·1 or 0·5% by weight. *G. C. Pether.*

Heat Stroke. A Clinico-Pathologic Study of 125 Fatal Cases. MALAMUD, N., HAYMAKER, W., and CUSTER, R. P. (1946). *Mil. Surg.*, **99**, 397.

The condition described is that resulting from the failure of the heat-regulating mechanism to deal effectively with increased production of heat due to a combination of excessive muscular exertion, high environmental temperature, and, in some cases, lack of acclimatization. All cases were in soldiers in training in the Southern States of the U.S.A. during the summer months. Clinically the onset was acute in 71%, sub-acute in 21%, and insidious in 8%. Death occurred, usually in coma, within 24 hours in 70% of patients, and in 1 to 12 days in the remainder. Where bronchopneumonia supervened there was no coma. Of the pathological changes, those in the brain were the most important ; cerebral œdema and congestion, and progressive neurone degeneration were almost invariable, with glial proliferation in the longer-standing cases ; petechial hæmorrhages were common. Changes in the other organs were inconclusive and appeared to be those of shock and toxæmia, subserous and submucous hæmorrhages being frequent. Laboratory investigations before death showed a platelet deficiency in 6 cases tested ; in the cases in which death was delayed prothrombin times were prolonged. It is concluded that two factors appear to operate in heat stroke—namely, hyperthermia and shock. *W. S. Killpack.*

INDUSTRIAL LUNG DISEASE

Examination of a Case of Silicosis Occurring in a Stonemason. (Untersuchung eines Falles von Steinhauersilikose.) ALBERTINI, A. V., BRANDENBERGER, E., and RÜTTNER, J. R. (1946). *Z. Unfallmed. Berufskrankh.*, **39**, 361.

The authors describe the case of a stone-mason, aged 63, who had worked for 46 years with various kinds of stone containing quartz. Among these were the sandstones of the Palatinate and the Main Valley and also Grossmünster stone. The silica content of these ranges between 40 and 80%. It is considered that the silicosis risk in this occupation is comparatively small, this being attributed in part to the large average size of the dust particles. The patient first noted symptoms in 1935—namely cough, dyspnœa, fatigue, and loss of weight. A radiograph was taken a year later and showed fine mottling of both lungs. No tubercle bacilli were found. As a result of this examination the patient was given a 100% disability pension. In 1943 he had signs of myocarditis and arteriosclerosis. Some doubt was cast on the original diagnosis, and it was noticed that the mottling was most marked in the upper zones. The lesions were calcified and there was also some pleural thickening. In 1945 the patient developed an œsophageal cancer from which he died.

At necropsy the nodules in the lungs were found to be of a greyish colour, uniformly distributed, and up to 5 mm. in size. The lymph nodes around the bronchi and trachea, in the aortic region, and around the pancreas were enlarged and hard and contained scattered white nodules of mineral matter. Histological examination of some of these revealed numerous particles of coal dust and shiny, needle-shaped crystals, later identified as lipoid crystals. In some places where the nodules in the lungs were near the surface the pleura was thickened and adherent. The lungs were partly collapsed and showed emphysematous areas. By a special technique the authors were able to demonstrate the presence of quartz in the hilar and abdominal lymph nodes. Hydroxyl-apatite was found in the lungs and hilar nodes, calcium phosphate in the hilar nodes, and magnetite in the lung tissue.

The authors note the surprising fact that in the old silicotic nodules of the lungs, demonstrated histologically, no quartz could be found, but where the lesions

were active and reaction most violent, as in the hilar and abdominal nodes, quartz was identifiable. The findings support the views of Albertini that the quartz particles are gradually transported from the lung to the hilar nodes and thence to the abdominal nodes. The mineral particles are in a fine state of division and suspension, and are possibly broken up further in their passage. The authors reject the view that silica is dissolved and then precipitated, because it always comes down in the amorphous form when this occurs. They warn against identifying all crystals as of mineral origin, since many are organic and lipoid. In the patient described the appearance of hydroxyl-apatite together with calcium phosphate in the hilar nodes must be regarded as unusual since the former generally occurs alone. The authors do not think that the silicotic condition had anything to do with the development of the œsophageal carcinoma. *G. C. Pether.*

Sedimentation Rate after Effort in the Diagnosis of Silicosis. (La velocità di sedimentazione dopo sforzo nella diagnosis della silicosi.) PARMEGGIANI, L. (1946). *Med. d. Lavoro*, **37**, 216.

Owing to the difficulties sometimes encountered in the diagnosis of the presence of tuberculosis in silicotics, the author tried to find out if the erythrocyte sedimentation rate readings before and after effort would help. The method used was to estimate the E.S.R. in the morning on a fasting and resting patient. Then it was repeated after a standard exercise (still fasting). The exercise was the ascent of 60 stairs as fast as the patient's condition would allow. Those incapable of doing this were asked to go for a 15 to 30-minute walk. The test was performed on the following groups of patients: (*a*) uncomplicated reticular silicosis, 8; (*b*) confluent silicosis, 15; (*c*) silicosis with inactive (?) tuberculosis, 11; (*d*) silicosis with active tuberculosis, 10; (*e*) silicosis with unassociated disease, 6; (*f*) healthy controls, 20. Group (*e*) included those patients with pleural calcification and thickening, apical sclerosis, discrete nodularities in the lung fields, lobar fibrosis, and peribronchitis. Group (*e*) consisted of 2 cases of acute bronchitis and 1 each of cardiac failure, syphilis, post-traumatic pneumothorax, and minor injury.

The results showed that after the test effort, in groups (*a*), (*e*) (except for the 2 cases of acute bronchitis in the febrile stage), and (*f*), the E.S.R. was unaltered. In group (*b*) it was increased by 7 to 35%. In group (*d*) it was constantly increased by 30 to 70%. In group (*c*) it was raised in some cases and unaltered in others. More careful investigation later proved that those patients in group (*c*) whose E.S.R. was raised by effort did actually have active tuberculosis. From these findings the author claims that the E.S.R. estimations before and after a standard exercise are of value in deciding, in conjunction with other signs, upon the activity of a tuberculous lesion in silicotics suspected of infection. *Tom Rowntree.*

The Determination of Free Silica in Industrial Dust. DURKAN, T. M. (1946). *J. industr. Hyg.*, **28**, 217.

Quartz is the most common form of free silica encountered in industrial hygiene work, and is usually the only one that need be considered in investigating a siliceous hazard. The numerous chemical methods of analysis hitherto used are described at some length, while petrographic methods, the *x*-ray diffraction method, and differential thermal analysis are described more briefly. For several years free-silica determinations have been made at the Saranac Laboratory, New York, by a method applicable to a great variety of materials, such as atmospheric and settled dusts, ore and rock samples, and mineral residues of tissue. The sample is treated first with hydrochloric acid to remove easily soluble compounds, then with phosphoric acid to break down silicates, and finally with dilute hydrofluoric acid to dissolve colloidal silica liberated by the other acids. A small portion of the residue which survives this treatment may be subjected to petrographic or *x*-ray diffraction examination; the remainder is analysed by standard methods for its total silica content. From the data obtained the percentage of free silica can quickly be calculated. For details of the analytical methods employed the original paper should be consulted. It also includes details of the results obtained on treatment of various silicate minerals, various kinds of quartz, and of synthetic mixtures of powdered quartz and other minerals prepared to simulate common industrial dusts. *H. M. Vernon.*

Study of the Regulations concerning Compensation in cases of Silicosis. (Etude de L'ordonnance relative à la reparation de la silicose.) MAZEL, P., and ROCHE, L. (1946). *J. Med. Lyon*, **27**, 63.

Silicosis used not to be regarded in France as a professional disease, and did not entitle workers to compensation. The Regulations of Aug. 2, 1945, which have been in force since Feb. 2, 1946, fill this gap. The authors inquire into the rules governing compensation for silicosis, their modifications, and the difficulties of their practical application. *L. Roche.*

Tuberculosis in Industry. HEAF, F. (1946). *Brit. med. J.*, **2**, 975.

Occupation, while it may predispose workers to tuberculosis, seldom causes it. The staff of sanatoria are exposed with impunity to highly infectious cases. The prevalence of tuberculosis among the middle aged and among males today is probably due to the heavier work carried out by the male. In industries where there is a special risk from silica dust the mean age of the tuberculous at death is higher than that of the tuberculous in other occupations. Apart from such specific risk, poor diet, over-work, anxiety, overcrowding, and poor ventilation are probably the chief predisposing causes. Dusts may increase catarrh and carry infection. Light work under bad conditions may be more harmful than heavy work under good conditions, as is seen by comparing the incidence among hairdressers and clerks with that of farm workers. The graphs shown in this paper of the age distribution of mortality from tuberculosis in various occupations and social classes are based on the years 1931–2 and are now out-of-date. Today, with the help of mass radiography, the industrial medical officer may be able to detect early cases, but he is then faced with difficult problems concerning the suitability of these patients for employment. If they are infectious, they should be placed under treatment; if the work exposes them to risk from injurious dust, they must stop such work. Many will be closed cases, and the question will arise whether the patients should be permitted in their own interests to continue at work. Each case must be dealt with individually, according to the nature of the occupation and the general health of the worker. Rehabilitation must be considered, and also the pro-

visions of the Disabled Persons Employment Act. A national tuberculosis research organization for assessing disability would be valuable. *E. L. Collis.*

Respiratory Air Flow Characteristics and their Relation to Certain Lung Conditions Occurring in Industry. SILVERMAN, L. (1946). *J. industr. Hyg.*, **28**, 183.

The author, in conjunction with Lee and Drinker, recently described a portable instrument for measuring the instantaneous rate of air flow during respiration without offering a significant resistance to air flow. The recording instrument for inspiration consists of a fine platinum wire suspended across the tube through which the air flows. The wire is pivoted at one end and connected with a spring at the other, and it is deflected in direct proportion to the rate of air flow. The deflection is photographed on a moving-paper camera. The expiratory instrument is of similar construction, but is provided with a heating bath to prevent condensation of exhaled moisture. For further details the original articles should be consulted.

This paper gives numerous samples of the records obtained from normal subjects and from 5 patients with asthma, 8 with pulmonary tuberculosis, 19 with silicosis, and 3 with primary emphysema. Tuberculous patients with recently developed fibrotic tissue show a positive result, as the bronchial constriction causes a smoothing and damping of the air-flow curve, particularly in the expiratory phase of the cycle. Loss of elasticity in the lung, due to emphysema, also produces a damping of the air-flow curve. No appreciable change in the shape of the curve was noted in the first or second stage of silicosis, but advanced second and third stage cases showed a marked damping. The method appears to be of value in distinguishing between lung depositions such as silica, which cause fibrosis, and those such as iron fumes, which accentuate the *x*-ray appearance but do not produce any disabling fibrosis. *H. M. Vernon.*

INDUSTRIAL DERMATITIS

Teak-wood Dermatitis. (Sulla dermatosi professionale da legno di teak.) DELOR, F. (1946). *Med. Lavoro.*, **37**, 244.

An oily substance, and a resin soluble in alcohol, ether, and chloroform have been extracted from teak. These, as shown in skin-sensitivity tests, may be irritant to certain persons. It is suggested that the irritating powers of the resin are due to a high content in unsaturated acids. For tests to be reliable the wood samples used must be fresh. Splinters of wood may cause irritation. Extracts made in chloroform, alcohol, ether, benzol, oil, and water give cutaneous reactions of decreasing intensity. Although the evidence adduced seems convincing, the author inquires whether the resinous substances are alone responsible for the reactions seen. For if this were so one could not explain why aqueous extracts may contain an irritant. He concludes that several irritants may be present, and not only in the resins.

A patient of the author's, aged 47, a carpenter, a few days after his first contact with teak developed irritation and erythema of the forearms. This cleared after a few days' rest but reappeared when he again used the wood, the face becoming involved and the rash more severe. When his condition improved, skin tests were made with the wood dust and with watery and alcoholic extracts, and these gave positive results. The skin eruption cleared with simple remedies.

The author investigated the possibility that the irritant is an alkaloid. An alcoholic extract of wood dust was made and treated with lime. Chloroform was then added, and acidified water in which the harmful substance was sought. Phosphotungstic acid was used as an indicator, but results were negative. Negative results were also obtained in a colorimetric test for saponins, with a concentrated sulphuric acid extract. The author suggests that the harmful substances are multiple, that the mineral salts cannot be ignored, and that repeated contact may neutralize the normal acid reaction of the skin. This leads to weakening of the keratin layer. *G. C. Pether.*

Contact Dermatitis : An Analysis or Tabulation of all Cases Proved in a Single Year. HOWELL, J. B. (1946). *Arch. Derm. Syph., Chicago*, **53**, 265.

The importance of contact irritants employed as topical medications or encountered as occupational hazards is discussed. All 250 cases were proved by patch tests. Topical applications were the commonest cause of contact dermatitis; sulphonamides and mercurials being the most potent sensitizers. Warning is given of the danger that patch tests with sulphonamides may photosensitize the patient or cause severe aggravation of the existing eruption; small test doses given orally are preferred to confirm sensitivity.

Of 86 cases of contact dermatitis of occupational origin, 23% occurred in the aircraft industry from formaldehyde and phenol formaldehyde (for plastics), glue, and rubber gloves—zinc chromate primer being the commonest sensitizer.

Attention is drawn to the importance of oleoresins in weeds as an occupational hazard to farmers and outdoor workers—35 (41%) of the total occupational cases being so caused.

Weed dermatitis usually begins in the spring on the ankles, later affects other exposed parts, and recedes in winter, but recurs more severely each succeeding spring. Polyvalent sensitization is the rule. Two forms of management of such dermatitis are advocated: (*a*) absolute avoidance of vegetation; (*b*) oral administration of specific weed oils during winter months.

Nickel is often the sensitizing agent in cases from jewellery or wearing apparel; in the list of 58 cosmetics implicated, 20 occurred from nail polish and 21 from hair lacquer. *Geoffrey A. Hodgson.*

ACCIDENTS AND ORTHOPÆDIC SURGERY

Industrial Injuries due to Noise and their Prophylaxis. (Über Lärmschädigungen in industriellen Betrieben und ihre Verhütung.) RUSSI, U. (1946). *Pract. otorhino-laryng.*, **8**, 71.

Investigations were carried out in a boiler-making shop, a stamping mill, and a weaving mill. The first batch of 8 workers comprised 3 from a rolling mill and 5 from a boiler-making shop. Sound-spectrum analysis in proximity to the pneumatic chisel gave a maximum of 115 phons with the peak between 600 and 2,000 cycles. All workers showed the characteristic drop at 4,000 cycles in their audiograms, the loss being greatest and most generalized for those with the longest period of employment.

The second group comprised 7 workers employed in a stamping mill. All showed the characteristic loss at 4,000 cycles. The third group contained 8 weavers. The noise level in the mill was such that the weavers could hear each other only if they shouted at close

range. All 8 weavers showed the typical 4,000-cycle drop in the audiograms. In those with the longest period of employment there was a high-tone falling curve, inability to hear a whisper, and tinnitus.

The second part of the investigation was carried out among the weavers, who were tested on Friday evening after a 48-hour week, on Monday morning after a 60-hour rest, and again on Monday evening after a working day. All showed improvement after their rest, this being greater in those whose employment had been of shorter duration. Complete relapse occurred by Monday evening. Four people with normal hearing were then subjected to a 2-hour period in the weaving mill. The whispered voice was heard at 2 to 5 metres (instead of 50 metres before entering the mill). Their audiograms showed the typical drop at 4,000 cycles. They complained of tinnitus, tiredness, desire to sleep, and in some cases a feeling of pressure in the head and giddiness. The test of 2 hours in the weaving mill in 4 normal controls was repeated with earplugs inserted. No hearing loss was recorded. The same test was carried out on 2 people with normal hearing, earplugs being put in the right ear only. The right ear was completely protected, but the left showed the usual changes. Finally 8 weavers, tested after a 60-hour rest period, wore earplugs on return to work. The recovery which had occurred during the rest period was maintained after a full working day.

[This is a comprehensive investigation. The literature is amply dealt with, but there is no reference to the work of Dickson and others in this country in 1939 on the effect of aircraft-engine noise.] *Stephen Suggit.*

The " Last-straw " Factor in Low Back Disability. With Summary of 100 Cases Examined and Evaluated by the Medical Advisory Board of the Industrial Commission of Arizona, 1934–1943. WATKINS, W. W. (1947). *Radiology*, **48**, 20.

This report is based on 100 cases examined and evaluated by the Medical Advisory Board of the Industrial Commission of Arizona in 1934–43. The author points out the difficulty in assessing the compensation for injuries, alleged or real, sustained by workers whose radiographs immediately after an accident show evidence of developmental anomalies or pathological changes. He considers that, while such conditions as rheumatoid arthritis produce clinical signs before radiographic changes, osteo-arthritic changes are usually symptomless long after they are radiologically evident. A workman with either of these conditions who sustains an injury may well be disabled for a very long time, and serial radiographs show progressive development of the pathological processes. The assessment of compensation for this " last straw " damage is very much more difficult than is suggested by the awards of the Medical Advisory Board. *J. F. Brailsford.*

Use of Berman Locator in Removal of Magnetic Intraocular Foreign Bodies. GUY, L. P. (1946). *Arch. Ophthal., Chicago*, **36**, 540.

For the removal of a magnetic foreign body from the eye precise location of it is necessary, since the application of a magnet may fail if the incision is even 1 mm. away from the site. Ordinarily *x*-ray examination will disclose the presence of a foreign body, and also its size and approximate position. However, *x*-ray examination may not be available, or the radiographer may not be skilled in special ophthalmic technique.

At the New York Eye and Ear Infirmary the Berman locator has been found helpful; its use in location is simple, direct, immediate, and of pin-point accuracy. Its application is readily mastered by an ordinary surgical staff. It is a portable electromagnetic device, similar in size and appearance to a wireless set. It has a sensitive probe holding a detecting element small enough for intraocular foreign-body work. The changes in the field caused by the approach of a metallic body register on an indicator, where it can be seen, or the changing pitch can be heard on the built-in loud-speaker.

The tip of the probe is applied first to the closed lid; if a ready response is obtained the search is continued by moving the probe along the surface until the " peak indication " occurs. If no immediate response is obtained, the probe is pressed into the tissues with the forefinger, the pressure being made and relaxed in a regular rhythm; if a foreign body is within range beneath the surface the indicator will respond. If there is no response the magnetic foreign body must be out of range of the surface, and initial exposure must be made of the globe, with the aid of radiology alone, and the search made as before. Whenever the " peak indication " is obtained the probe is held against the lid or the globe at that point and the patient is instructed to open his eye and rotate it in various directions; a consistent change in response to the movements of the globe indicates that the foreign body is either intraocular or in tissues which move with the eye; if there is no such change the foreign body is extraocular. When the foreign body, after having been located, changes its position, the new position can be recognized easily; the incision into the globe is made just beneath the centre of the probe where the " peak indication " was obtained.
D. Matheson Mackay.

ENVIRONMENT

Sanitation for Food Departments. MALLMANN, W. L. (1946). *J. Amer. diet. Ass.*, **22**, 870.

In 1943 the United States Public Health Department reported epidemics of dysentery, chemical and food poisoning, gastro-enteritis, typhoid, paratyphoid, scarlet fever, trichinosis, and botulism, all of which were carried by foods. There were numerous outbreaks of trench mouth, septic sore throat, and common cold, spread by improperly washed dishes. As a result of experiments it was concluded that the temperature of the washing water should be between 130° and 140° F. and that of the rinsing water 170° F. Dishes should be immersed in the rinsing water for not less than 10 seconds. Perishable goods should be stored at temperatures below 50° F., and cooked foods, such as custards and salads, should not be allowed to cool at room temperature before refrigeration. *Geoffrey McComas.*

Food Utensil Bacteriology. HUTCHINSON, I. (1947). *Brit. med. J.*, **1**, 134.

Utensils in 25 kitchens of hotels, school canteens, and snack bars, were examined. Samples of washing-up water were taken, and swabs moistened in nutrient broth employed for cultures from spoons, cups, forks, glasses, and plates, which had been used, washed, and stacked for re-use. Coliform organisms were found on 21% of implements; coagulase-positive staphylococci on 5·5%; hæmolytic streptococci on 2·3%, the most frequent isolations belonging to groups B and G. A group A strain was found once only (on a fork). Other organisms found were *Bacillus fæcalis alkaligenes*, " paracolon bacilli," *Proteus*, *Pseudomonas pyocyanea*, and, once, a

Sonne dysentery organism. Fæcal streptococci, *Streptococcus viridans*, and *Staphylococcus albus* were found on most implements.

Examinations were made of 38 samples of dish-washing water, of which 19 had counts of more than a million organisms per c.cm. at 37° C., and 9 of more than a million organisms per c.cm. at 22° C. The ratio of counts at the two temperatures suggested animal origin rather than soil or dust, and coliform counts were high. The water was thus bacteriologically similar to sewage. In 11 samples there was no soap or soda. The value of soda is indicated by the fall in the number of specimens with high total and coliform counts when the *p*H was over 10 as compared with a *p*H range of 7 to 10. The temperatures of water—average 40° C., and maximum 50° C.—were found to be too low to have any bactericidal effect.

The cleanest utensils were found where a washing machine with soda was used and a drying oven took the place of hand drying—half the swabs being sterile. Drying-cloths were also examined; 17 of 36 yielded only fæcal streptococci, *Staph. albus*, *Str. viridans*, and spore-bearing aerobes. The others yielded one or more of the following: coliform bacilli, paracolon bacilli, *Staph. aureus*, and *B. fæcalis alkaligenes*. Sanitary arrangements and washing facilities for the staff were in most cases unsatisfactory. *G. T. L. Archer.*

Dishwashing Practice and Effectiveness (Swab-rinse Test) in a Large City as Revealed by a Survey of 1,000 Restaurants. KLEINFELD, H. J., and BUCHBINDER, L. (1947). *Amer. J. publ. Hlth.*, **37**, 379.

The first of these investigations deals with the technique of "swab-rinse" tests of utensils in restaurants, and is of interest to those using such a method. The second gives the results of a survey of 1,005 restaurants in New York City. Hand washing of utensils was practised, wholly or partly, in 88% of these, and machines were used in 17·5%. In the establishments where utensils were washed by hand only 10·5% of glasses and cups reached a satisfactory standard of cleanliness; even in the small number of restaurants where mechanical methods were used only 35·8% reached the standard. Better figures were obtained with spoons and forks, and there was little difference between those washed by hand and those washed by machine. In cafeterias the standard of washing, whether by hand or by machine, was inferior to that in "waiter service restaurants." *C. O. Stallybrass.*

The Cleansing and "Sterilization" of Mess Tins in the Field. MANN, B. (1946). *Publ. Hlth., Lond.*, **59**, 161.

The danger of spread of infection by mess equipment arises partly from cross-transmission of nose, throat, and intestinal commensals when mess tins are immersed in a common rinse, and partly from fly-borne spread when the tins are left exposed between meals. Organisms which have been isolated from eating utensils include *Staphylococcus albus* and *aureus*, *Escherichia coli*, hæmolytic streptococci, pneumococci, and diphtheroids. The efficient sterilization of equipment in barracks, where washing and drying machines, cleansing powders, and detergents are available, presents little difficulty. The problem confronting a mobile field force using army-pattern mess tins is more formidable. The basis of most practices is the swilling of the utensils in a container of water. Any sterilizing action is expected to result in some cases from the heat of the water, in others from an added chemical.

Investigations proved that though low-temperature rinses with cold hypochlorite solution might be effective in sterilizing comparatively clean utensils, greasy mess tins were not so much as cleansed, even at temperatures up to 50° C.; the free-chlorine content of the hot hypochlorite solution in the presence of organic matter very rapidly fell to negligible amounts. As to the effect of heat alone as a sterilizing agent, although a 5-second contact with water at a temperature not less than 80° C. resulted in the sterility of utensils contaminated by *E. coli*, this procedure was not sufficient in the case of tins impregnated with *Staph. aureus*, the organism most resistant to heat. Even a double rinse of 5 seconds' duration, the first in hot water at 80° C., the other in cold hypochlorite solution, failed to destroy this organism. A double rinse, each of 5 seconds' contact in hot water at 80° C., was almost always completely successful. As an indicator that the water was at 80° C. a small phial containing 2 g. of naphthalene and a crystal of brilliant green were used. Below 80° C. the naphthalene is solid, but at this temperature it liquefies.

A suggested drill for field sterilization of mess tins is that at the end of a meal troops rinse their dirty mess equipment in water at or above 80° C., the maintenance of this temperature being shown by the immersion indicator. This single rinse will destroy all *E. coli*, all hæmolytic streptococci, and most of the *Staph. aureus*. On entering for the next meal, troops again rinse the mess equipment in a drum containing water at not less than 80° C. This second rinse will destroy all the remaining *Staph. aureus* and organisms resulting from any contamination of flies which might have occurred. The second rinse, incidentally, warms the containers—an advantage when hot meals are being served.

[Experiments are urgently required to determine the measures necessary to destroy the viruses of infective hepatitis and poliomyelitis.] *Caryl Thomas.*

Use of "Gammexane" Dust Against Domestic Cockroaches. HYNES, H. B. N. (1947). *Nature, Lond.*, **159**, 200.

Almost all the houses in Mogadishu (capital of former Italian Somaliland) are seriously infested with cockroaches. Spraying with pyrethrum or D.D.T. having produced poor results, it was decided to attempt control with "gammexane" dust containing 10% "active ingredient," which was applied with a simple hand-duster. The author states that the dust did not affect the eggs and that it was advisable to dust again after an interval of 3 or 4 weeks. Provided these two dustings were carried out, using about 250 ml. of dust for each treatment of a five-roomed house, the premises so treated remained reasonably free from cockroaches for about 3 months, after which time they were gradually re-invaded and treatment had to be repeated. *R. M. Gordon.*

Tsetse Fly Repellents. FINDLAY, G. M., HARDWICKE, J., and PHELPS, A. J. (1946). *Trans. R. Soc. trop. Med. Hyg.*, **40**, 341.

The authors began their experiments by testing, in the field, the repellent properties of the following nine substances or compounds against *Glossina*: (1) *N*-sec-butylphthalamide; (2) 2-phenylethyl-α-hydroxyisobutyrate; (3) *N-n*-amylsuccinimide; (4) benzyl ether; (5) 2-phenyl-cyclohexanol; (6) *n*-butyl-*dl* malate; (7) ethylhexane-diol-1,3 (Rutger's 612); (8) 2,2'-dimethyl-2-carbobutoxy-dihydro-7-pyrone (indalone); (9) Formula 622 (dimethyl phthalate, 6 parts; indalone, 2 parts; Rutger's 612, 2 parts). The result of this preliminary

screening proved that indalone and Formula 622 gave the most striking results, and in consequence the following additional tests were carried out with these compounds, controls being employed in each observation. (1) Duration of effectiveness of indalone and Formula 622. To determine the time during which these compounds were effective they were applied at intervals of from 7 to 8 hours before exposure to tsetse bites. Both compounds showed a loss of efficiency between 4 and 6 hours after application. (2) The effect of sunlight on the efficiency of Formula 622. Two treated and 2 control fly-boys sat for 30 minutes exposed to the direct rays of the sun in a tsetse-free area. They then sat for 1 hour in a shaded tsetse area. Some loss of repellent power was noted under these conditions. (3) The effect of sweating on the efficiency of Formula 622. Two treated and 2 untreated fly-boys danced strenuously in the shade for 30 minutes. They were then exposed to tsetse flies for 1 hour. No loss of efficiency as the result of sweating was noted. *R. M. Gordon.*

Insect Tests of Wire Screening Effectiveness. BLOCK, S. S. (1946). *Amer. J. publ. Hlth.*, **36**, 1279.

The author describes experiments with mosquitoes and other insects to test the effectiveness of wire screening with square and rectangular apertures. The screening tested was made of wire of 0·011 in. diameter, but the meshes investigated were 18×18, 16×16, and 14×14 for square meshes and 18×16, 18×14, and 18×12 for rectangular meshes. The majority of the tests were carried out with laboratory-bred mosquitoes—*Anopheles quadrimaculatus*, *A. quinquefasciatus*, and *Aëdes aegypti* —in cages made of the screening under test. None of the meshes permitted the escape of *A. quadrimaculatus* or *A. quinquefasciatus*, but undersized *Aëdes aegypti* escaped through all the meshes and normal-sized *A. aegypti* through most. The percentage of *Aëdes* escaping was found to be directly proportional to the length of the diagonal of the apertures. A mesh of 18×14 proved as effective as one of 16×16. The placing of the rectangular aperture with the long axis vertical was important, since in 18×12 mesh a greater proportion of *Aëdes* escaped when the long axis was horizontal. The effectiveness of different meshes against small light-attracted insects (gnats, tiny moths) was studied, and in these also the proportion of penetrations was directly related to the length of the diagonal of the aperture. Only 18×18 or finer mesh acted as an appreciable barrier to these small insects.

[This is a careful study of the comparative effectiveness of wire screening with the rectangular or the square apertures. The former is more rapidly woven and should therefore be cheaper. No reference is made to British literature on the greater importance of aperture measurements as compared with mesh counts.] *T. H. Davey.*

Developments in Disinfestation of Ships. CARGILL, W. P. (1946). *Publ. Hlth., Lond.*, **60**, 16.

This paper draws attention to the value of D.D.T. and "gammexane" in insect infestation on shipboard. Gammexane, the gamma isomer of benzene hexachloride, is both reliable and persistent, but suffers from a disagreeable odour due to impurity: the powder D.034 contains 20% crude gammexane. This powder is applied with a blower into crevices where the dust will remain active for several months. A purer form in solution in kerosene will, it is stated, be available shortly.

D.D.T. has a faint smell; it is insoluble in water but soluble in kerosene. In ships it is commonly used as a 5% solution in kerosene and is applied by hand or a power spray; there is risk of fire from the use of sprays, and full precautions should be taken until the spray has dried. D.D.T. is effective as a 5% powder, though in the U.S.A. 10% is preferred; it can be applied by a blower—either hand-blown, or automatic as in the "Freon" bomb. Both gammexane and D.D.T. are effective under practical conditions against bugs and cockroaches as well as lice; both approach the ideal as insecticides. *C. O. Stallybrace.*

GENERAL

Investigations into the Fitness for and Prospects of Work in Male Diabetics. (En efterundersøkelse av mannlige diabetikere, med henblikk på arbeidsførhet og arbeidsmuligheter.) ELSTAD, D. (1946). *Nord. Med.*, **31**, 1807.

In 1940-41 a follow-up review was carried out of 283 men who had been treated for diabetes between 1930 and 1939. Replies to a questionary were received from 154 (65%); 12 (5%) had died in the interval. The average age was 38, and the duration of disease varied from 2 to 18 years; in more than half it was greater than 7 years, with an average of 7⅔ years. Most cases were fairly severe, 87% requiring more than 20 units of insulin daily, with a daily average for all cases of 55 units. A table shows the types of occupation pursued; one-half at least could be classed as heavy workers, but in most cases working hours and meal times were regular.

The replies showed that 87% (134) considered themselves completely fit for work and were continuing in their previous occupations; 11 (7·2%) were partially fit, having been forced to change to a more suitable occupation; while 9 (5·8%) reported that they were unfit for work. Three of these last cases of disability were not entirely due to diabetes, but partly to epilepsy and tuberculosis, 3 had cataract, and the remaining 3 could give no satisfactory reason for their incapacity. Patients were asked if they had adjusted themselves to a state of chronic illness, or if they felt it a hindrance in daily life; 111 (72%) had grown accustomed to their disability, while 34 (28%) felt that their disease was a hindrance, mainly on account of difficulties of diet. Of the total who replied 42% had had to call in medical assistance several times a year after discharge from hospital, 34% did so less frequently, and 24% were able to control the disease without outside aid.

D. J. Bauer.

PROCEEDINGS OF THE ASSOCIATION OF INDUSTRIAL MEDICAL OFFICERS

FORTY-EIGHTH MEETING

The Forty-Eighth Meeting of the Association of Industrial Medical Officers was held at Leeds on May 29, 30, and 31, 1947, Dr. W. Blood (London) being in the Chair.

Private Business

The Private Business Meeting was held on May 29, 1947, at the Algernon Firth Institute of Pathology, Leeds.

British Council for International Congresses Concerning Health and Safety

The following were appointed to represent the Association on the above Council until the Annual General Meeting, 1948: the Chairman and the Hon. Secretary (both ex officio), Dr. M. W. Goldblatt, and Dr. T. A. Lloyd Davies.

Central Council for Health Education

Dr. J. A. A. Mekelburg (London) was elected to serve on this Council as the representative of the Association.

Alteration of Constitution

A new Constitution was unanimously adopted. Under this the general management of the Association was entrusted to a widely representative Council presided over by a President. The Chairman expressed the view that by this means the Association would have more time to devote to technical meetings. He also referred to the fact that a considerable part of the business which had previously been transacted by the Association would now be taken over by the Industrial Medicine Committee of the British Medical Association. It was further agreed that the present Executive Committee should act as a "Caretaker Council" until elections to the Council had taken place.

Election of Members

Seven Ordinary and eight Associate members were elected.

Public Business

On Friday, May 30, the morning was spent visiting the Yorkshire Copper Works Limited in Leeds. This particular works is the largest of its kind in the United Kingdom. An explanatory lecture, aided by the epidiascope, gave the members a preview of the tour which followed and added greatly to the interest of the visit to the various parts of the works. Guides from the staff conducted the parties round, and explained the processes which changed the initial crude material by many stages to the finished products. The works cover nearly fifty acres of ground, and here during the late war over 29,000 miles of copper tubing were supplied for aircraft alone. Of particular instruction and interest were the processes associated with the moulding of the billets and their subsequent transformation into tubes.

The tour included a visit to the medical section, under the guidance of the medical officer of the works.

The Managing Director was present at a buffet lunch at which members were introduced to the local Trades Union representative, who had been at the works for many years. Addresses of appreciation were made by the works medical officer and the Chairman of the Association, and were responded to by the Managing Director and the Trades Union representative.

In the afternoon a visit was made to Imperial Chemical Industries Limited, Dyestuffs Division, in Huddersfield. The party was divided into four groups and conducted round the various sections of the works. Included in the tour were the departments associated with the manufacture of nylon, "agrosan," and aniline, the standardizing department, where an exhibition of colours and pharmaceutical products was seen, and various nitration processes which are carried out in the primaries area. On the non-manufacturing side the visits included the joiners' shop, the boiler plant, and the power house. In spite of an accompanying thunder-storm, the tours were, in the main, carried out in full and the visitors, most of whom had not seen such a works before, were impressed by the size and complexity of the various manufactures.

Representatives of the management accompanied the party to the recreation club and joined in tea. In the tea room was an interesting exhibition of safety appliances and protective clothing, which were demonstrated by live models.

The Association Dinner took place on the evening of Friday, May 30, at the Mansion Hotel, Roundhay Park. The guests included Dr. J. T. Ingram, Professor M. J. Stewart, and Professor F. S. Fowweather. The toast of "The King" was given by Dr. Blood. The health of the Association was proposed by Dr. Ingram and replied to by Dr. Blood. The health of the guests was proposed by Dr. A. F. Shirras, and replied to by Professor Stewart.

A clinical meeting was held on Saturday morning, May 31. Dr. J. T. Ingram took as his subject the industrial dermatoses. These he classified as: (1) those peculiar to a particular trade or occupation; (2) traumatic, where the manifestations of trauma may show themselves in any case of contact; (3) eczematous, occurring in the minority of workers and associated with (*a*) contact, with resultant developed sensitivity, and (*b*) wear and tear, as in washerwomen; (4) dermatitis arising in the eczematous-prone.

In discussing diagnosis, Dr. Ingram pointed out how essential it was to examine the stripped patient and to be particularly watchful for scabies, to examine the feet for ringworm, pompholyx and fungus infections, and the legs for gravitational phenomena, and to note any abnormality of the skin of the thighs. Careful case-taking history was most important, and the site of affection and course should be carefully studied for differential diagnosis. Industrial dermatitis tended to affect the exposed parts (except perhaps the palmar surfaces of the hands), and sites where opposing surfaces and moisture are present such as the flexures of the arms and the axillæ. Patch tests, when carried out carefully and correctly, were of great value in diagnosis. In treatment it was important to keep to simple technique—calamine lotion, Lassar's paste, and other simple applications. Penicillin ointments were not recommended.

Professor A. N. Shimmin later in the morning addressed the meeting on the industries of Leeds. He pointed out that among the 206,000 insured workers in the area,

wholesale clothing accounted for the largest group (34,859). This was followed by 24,884 in distributive trades, 23,127 in general engineering, and 10,653 in National Government services. Although Leeds was on a coalfield, coal had not dominated the industrial development of the city. Until the late eighteenth century the trade in wool and wool fabrics dominated the scene for three or four hundred years. In the early nineteenth century engineering took the lead, and now the making of clothes had taken first place. The rapid growth of the tailoring industry was explained by the advent of the sewing machine in the 1850's, the discovery of methods of using new and old rags as re-entrant material (shoddy and mungo) in the making of cloth, and the adaptation of the band-knife to the cutting in bulk of patterns for garments.

Leeds was far from being a one-industry town, however. Printing, chemicals, leather manufacture, confectionery, and a host of other employments maintained the cosmopolitan character of its growth. Its critics said that the range of industries had robbed Leeds of a central purpose, but the critics must remember that versatility was a great merit, and Leeds usually escaped the extremes of boom and depression just because its industries were so varied.

The Chairman moved a vote of thanks to the Leeds Group for a most enjoyable meeting; this was carried with acclamation.

NOTTINGHAM GROUP

The Annual General Meeting of the Nottingham Group of the Association of Industrial Medical Officers was held at the Black Boy Hotel on June 12, 1947. There were present Drs. Keatinge, Lloyd-Davies, Collis, Levin, Mayne, Shaw, Whimster, Spark, Crooks, and Young.

The accounts for the year 1946–47 were unanimously adopted.

Dr. G. E. C. Collis, Ransome and Marles Ltd., Newark, was elected Chairman, and Dr. J. Magill Young, Brush Electrical Engineering Co. Ltd., Loughborough, was appointed Hon. Secretary. It was unanimously agreed that Dr. Young be appointed as an Ordinary Member of the Council representing the Group in accordance with the new constitution.

The Meeting was followed by a dinner at which Mr. W. Rothera, the Coroner for Nottingham, was present as a guest. After dinner Mr. Rothera opened a discussion on "The Coroner's Point of View," which led to a lively debate in which all present took part.

OBITUARIES

N. HOWARD MUMMERY

By the death of Howard Mummery at the age of 70, industrial medicine has lost one of its pioneers. He trained at University College, London, and graduated M.R.C.S., L.R.C.P., in 1902, and was a member of the B.M.A. for over twenty-eight years. In the 1918 war he had the unusual experience of serving in both the medical branch of the Royal Navy and the Royal Army Medical Corps, where he reached the rank of Lieutenant-Colonel. In 1924 he was appointed whole-time medical officer to J. Lyons and Company, London, where he found an opportunity to plan and administer one of the first large factory clinics in the country. He had a passion for detail, and his organization at Cadby Hall was outstanding in its time. When the Association of Industrial Medical Officers was formed in 1935, Howard Mummery, recognized then as a leader in his branch of medicine, was unanimously elected its first chairman. Much of the success of this society is due to his untiring efforts. His tact in the chair, his strength of character—amounting often to paternal sternness—his wide knowledge of his subject, and his mastery of administrative method, did much to oil the wheels of early meetings when tenseness in discussion could well have caused disharmony. At the end of 1936 he resigned his appointment in industrial medicine and settled in St. Helier, Jersey. He looked forward to a quiet retirement, but his clinical urge persisted and within a few months he began consulting work in dermatology, a branch of medicine in which he was especially experienced. The whole of his social existence soon was to be disintegrated however, for he left the island only some hours before its invasion by the Germans in 1940. Return to England was rapidly implemented by a return to industrial medicine. At the Bristol Aeroplane Company and with other firms his experience was once more placed at the disposal of industry, this time as his contribution to the war effort.

But as the war drew to a close his health began to fail, and, once again, he retreated to Jersey where he died on July 20, 1947. Industrial medicine was enlarged in no small measure by his work and example. D.S.

JOHN CROSTHWAITE BRIDGE

By the death of Dr. J. C. Bridge on September 27, Industrial Health has lost one of its outstanding figures who contributed much to the development of this service. Born in 1877, the second son of the Reverend James Henry Bridge, he was educated at Carlisle Grammar School and the Middlesex Hospital, gaining an entrance exhibition. Qualifying with the English Conjoint Diploma in 1902, his subsequent academic achievements were successively the D.P.H. of Cambridge in 1906, the F.R.C.S.(Ed.), in 1910, Barrister-at-Law (Middle Temple) in 1913, and the M.R.C.P.(Ed.) in 1920.

After serving first with the Carnegie Institute, Dunfermline, then as Assistant Medical Officer of Health, Devonport, and then as Medical Officer of Health for Breconshire, he entered the Home Office in 1914 as H. M. Medical Inspector of Factories. Here he was to find his life's work, serving for 29 years in the Factory Department, the last sixteen years as Senior Medical Inspector in succession to Sir Thomas Legge. During the 44 years of service of these two men, this country led the world in the application of medical science and practice to the development of industrial health legislation, and they were successively the dominant figures in this work.

Bridge was eminently practical, skilled in codifying knowledge and experience. He was particular in his pursuit of truth, and insistent on accuracy in himself and others. He was a clear thinker and fast worker, and eagerly sought for new knowledge to apply to specific problems in industrial health and so to increase the efficiency of preventive measures. Bridge really knew industrial processes and the comparative hazards attached to different stages of them. Thus it was that his contribution to the progress of industrial health was cumulative and widespread. He held strongly that legislation concerned with prevention should go hand in hand with legislation relating to compensation, and here too, as Adviser to the Home Officer on medical aspects of the law relating to compensation, he left his mark. He was assiduous in identifying new occupational diseases and ensuring their consideration with a view to their inclusion in the schedule of diseases subject to compensation.

He was a member of the Departmental Committee of 1930 on extensions to the Schedule of Industrial Diseases, and of another Committee in 1935 on certain questions arising under the Workmen's Compensation Act. He was Chairman of the Departmental Committee on Medical Arrangements for the Diagnosis of Silicosis in 1928, a member of the Industrial Health Research Board, of the Industrial Pulmonary Diseases Committee, and of the Committee on Industrial Solvents of the Medical Research Council, and of many other Committees such as the Ministry of Health's Committees on the Causes and Prevention of Blindness (1922), on Cancer (1928–35), on Ethyl Petrol (1928), and the Advisory Departmental Committee on Anthrax (1935–46), and the Correspondence Committee on Industrial Hygiene of the International Labour Office from 1927. He was vice-president of the Section of Occupational Diseases at the Annual Meeting of the British Medical Association in Manchester in 1929 and of the Section of Public Health at the Centenary Meeting in 1932. A very early member of the Institution of Professional Civil Servants, he became a member of Council and twice held the office of Chairman from 1922–26 and again from 1930–31. After his retirement from the Factory Department, he became Chairman of the Association of Industrial Medical Officers and also Consulting Officer to Imperial Chemical Industries.

Although so active in public affairs, Bridge was inherently retiring and sensitive, but he had withal a most disarming and appealing charm of word and manner. His colleagues, especially the junior ones, knew that he kept of his best for them. He was never too busy to discuss knotty points, and they never forgot how doubts and difficulties would vanish under his skilled analysis. Those who sat at his feet have an abiding gratitude for his unobtrusive teaching and his touch of genius—the full force of which was apparent only in retrospect. And so throughout his service he commanded a happy crew in a happy ship, whatever the hazards of the voyage.

Dr. Bridge was made a C.B.E. in 1934 and appointed Honorary Physician to H.M. The King in 1937.

He married first in 1904 Bessie (died 1933), daughter of Gateward Coleridge Davis, and in 1935 Joan Phyllis, daughter of James S. Hann, who survives him with one daughter.

E. R. A. M.

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